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Editor-in-Chief : SIR NILRATAN SIRCAR, Kt.

Vol. III **April, 1934** **No. 8**

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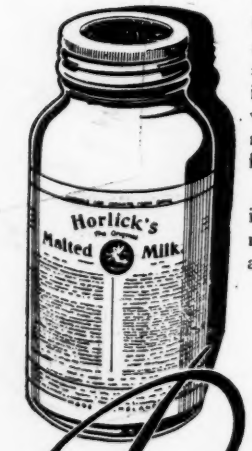

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JOURNAL
OF THE
INDIAN MEDICAL ASSOCIATION

Vol. III *Calcutta : April, 1934* No. 8

The Rationale and Results of the Treatment of Pulmonary Tuberculosis by Phrenic Exairesis—an Analysis of 286 Cases*

M. KESAVA PAI, M.D., K. VASUDEVA RAO, M.D., K. S. SANJIVI, M.D.
AND K. VENKAT RAO, M.B., B.S.

(*From the Government Tuberculosis Hospital, Madras*)

The value of *phrenic evulsion* in the more advanced cases of pulmonary tuberculosis, especially those where, due to pleural adhesion or activity in the collateral lung, artificial pneumothorax therapy is inapplicable, has now been firmly established as a result of the experience of the last 15 years and more. The old idea that phrenic exairesis was useful in basal lesions only is now known to be incorrect and the operation has been proved by practical results to be of benefit in apical lesions as well. In a paper contributed by two of us[1] to *Tubercle* in April 1932, we described the mechanism of respiratory movements and the effect of phrenic evulsion on these movements, clearly demonstrating by actual measurements that the diaphragmatic paralysis resulting from phrenic evulsion has a definite effect on both the basal and the apical regions of the lung, which accounted for the clinical observation that the operation benefited all cases irrespective of the seat of lesion. It was proved by measurements before and after phrenic exairesis that pulmonary collapse of a greater or less degree takes place at the base in 93% cases and at the apex in 74% and that a compensatory stretch of the lung takes place on the collateral side in about 50% cases both at the apex and at the base. WERNER and O'BRIEN[2] consider that the degree of collapse is greater in the right lung than in the left after phrenicectomy. The clinical results of phrenic exairesis in our cases were seen to correspond to the degree of collapse, 92% of the basal and 63% of the apical obtaining benefit, whilst 94% of the cases with involvement of the whole lung improved after the operation. The results obtained by COOPER[3] were similar. He observed that 42% of basal cavity cases and 30% of the apical cavity cases improve after phrenicectomy. The apparently smaller percentage in Cooper's cases was evidently due to the fact that it referred to the later results and not to the immediate as in our series.

In the present series of 286 cases a further confirmation of the observations made on the previous occasion has been effected. An attempt has also been made to classify cases according to the nature and extent of involvement and correlating the benefit obtained in the different types.

BRONFIN and CHERNYK[4] by an analysis of their cases have tried to explain why paralysis of the diaphragm benefits some types of cases more than others. They observe that decrease in the volume of the lung is very much greater than is apparent from the degree of rise of the dome of the diaphragm, a rise of 1 to 10 cm. effecting a decrease in the volume of the lung by fully $\frac{1}{4}$ to $\frac{1}{3}$. This contraction is progressive, taking weeks or even months to reach the maximum, being supplemented by a fibrosis resulting during the

*Paper read at the Twenty-first Indian Science Congress held at Bombay, January, 1934.

natural process of healing after the phrenicectomy. The extent of the rise of the diaphragm need not be proportional to the degree of contraction of the lung or the benefit obtained clinically, for several cases have been found where the immediate rise of the diaphragmatic dome was slight but the ultimate pulmonary contraction was marked and the clinical improvement considerable. LINDBERG[5] has recently proved by actual measurements that the secondary and tertiary rises of the diaphragm after phrenicectomy are often considerable in degree. Apparently the stoppage of the pumping action of the diaphragm leads to a series of processes by which not only is the progression of the disease controlled but progressive fibrosis of the lung is facilitated. The occlusion of the lymphatics and blood vessels, the increased control of the vagus and the sympathetic and the decrease of the movement in the downward direction are supposed to be mainly responsible for the progressive fibrosis and contraction of the lung.

The following are the various indications for phrenicectomy:

1. Unilateral lesions where pleural adhesion prevents the introduction of air into the pleural sac.
2. Bilateral cases with comparatively less collateral disease.
3. Cases of haemoptysis where pneumothorax collapse is not feasible due to adhesions.
4. Cases of partial induced pneumothorax where progress is unsatisfactory due to adhesions either at the base or at the apex, especially with a cavity still patent.
5. Cases of empyema and bronchial fistula.
6. Cases of bilateral disease too extensive to be treated by artificial pneumothorax.
7. Relief of symptoms caused by basal adhesions such as displacement of mediastinum, dragging pain, hiccough, dyspnoea etc.

IMMEDIATE RESULTS

The primary results of phrenic exairesis in our series of cases have been classified in the annexed Table I according to the extent and nature of the lung affection.

1. 79% benefited in unilateral involvement with apical cavitation, 38% showing considerable improvement.

2. 77% improved in unilateral involvement with cavities both at the apex and at the base, 44% receiving considerable benefit.

3. 66% benefited to a great extent amongst cases of bilateral involvement without cavitation.

4. 70% improved in bilateral involvement without cavitation on collateral side, 34% being considerably benefited.

5. 54% benefited in bilateral involvement with cavitation on both sides, 23% only receiving marked benefit.

TABLE I.

IMMEDIATE RESULTS

Nature and extent of lesion.	No. of cases.	Much Improved.	Improved.	No Result.	Died in Hospital.
1. Unilateral with upper zone cavity	29	11 38%	12 41%	6	...
2. Unilateral with basal cavity.	11	5 45%	5 45%	...	1
3. Unilateral with cavities in both zones.	36	16 44%	12 33%	8	...
4. Bilateral without cavitation either side.	3	2 66%	...	1	...
5. Bilateral without cavity on collateral side.	59	20 34%	21 36%	18	...
6. Bilateral with cavity on both sides.	124	29 23%	39 31%	50	6
7. Pyopneumothorax and Empyema	6	1 16%	4 66%	...	1
8. Non-tuberculous basal disease.	14	7 50%	5 36%	2	...
9. Chronic Pleurisy	4	3 75%	...	1	...
10. Artificial pneumothorax cases with collapsed cavities.	65	22 34%	30 46%	12	1

6. 90% of unilateral basal cavity cases benefited, 45% benefiting to a great extent.

7. 86% of cases of non-tuberculous basal disease including bronchiectasis, benefited, 50% showing great improvement.

8. 80% cases benefited when combined phrenicectomy and pneumothorax were adopted, 34% receiving marked benefit.

9. Empyema, pyo-pneumothorox and chronic pleurisy were also benefited in 75% to 82% cases.

Remote Results

Whilst the immediate results of phrenic evulsion in the advanced cases of pulmonary tuberculosis were gratifying the remote results were naturally much less so, as a large number either relapsed or grew progressively worse due to collateral extension after discharge from the hospital. Most of the cases were from amongst the illiterate lower classes whose whereabouts after discharge could not be traced. The results in table II are therefore of 67 cases only, classified according to the period after discharge from the hospital.

TABLE II.

Remote Results

Period after discharge from the hospital.	No. of cases.	Much Improved.	Improved.	No effect.	Died.
0— 6 months ...	41	15 36%	5 12%	2	19 46%
6—12 months ...	11	3 27%	1 9%	2	5 45%
12—18 months ...	6	...	3 50%	...	3 50%
18—24 months ...	5	3 60%	...	1	1 20%
24—30 months ...	4	1 25%	1 25%	1	1 25%
	67	22 33%	10 15%	6 9%	29 43%

It will be seen from the table that the mortality amongst the cases is about 50% during the first 18 months after discharge from the hospital. Of those that survived 18 months or more, the mortality was 25% or less. It is evident, therefore, that the benefit of phrenicectomy is due not so much to the primary collapse following the diaphragmatic paralysis, as to the progressive collapse secondary to the process of fibrosis resulting from the phrenicectomy. The longer the patient survives after the operation the greater the benefit. Graf's[6] results showed that 83·5% were living amongst 67 patients operated within 2 to 6 years previously.

References

[1] M. Kesava Pai and K. Vasudeva Rao: *Tubercle*, 295, April, 1932.

[2] Werner and O'Brien: *Jour. Roent. & Rad. Ther.* XXIV-626, Dec. 1930.

[3] Cooper: *Annl. Int. Med.* IV-1509, June, 1931.

[4] Bronfin and Chernyk: *Amer. Rev. Tub.* 689, Dec. 1932.

[5] Lindberg: *Amer. Rev. Tub.* 352, Septr. 1933.

[6] Graf: *Amer. Rev. Tub. Abst.* 35, Aug. 1932.

Radiography in Diagnosis of the Diseases of Alimentary System*

M. D. Joshi, M.B.B.S. (Bomb.), D.M.R.E. (Camb.)

Bombay

Necessity of X-ray in Diagnosis

Sometimes similar symptoms are produced by some entirely different pathological conditions and a mistake is likely to be made in the clinical diagnosis of such conditions. A patient who had clinical symptoms of gastric ulcer showed a stone of the kidney in X-ray examination. In another case a stone in right ureter at the level of the tip of the transverse process of the 5th lumbar vertebra gave typical symptoms of chronic appendicitis. Again sometimes it is very difficult to diagnose gall-stones, duodenal ulcer and appendicitis, from one another on account of reflex pain and similar and deceptive symptoms. When a tumour is felt in right hypochondriac region it is not infrequently difficult to decide whether the tumour is in connection with kidney, liver, gall-bladder, pyloric end of stomach, head of pancreas and ascending colon. It is the X-ray that can give us some reliable information in such cases.

The Technique

The diagnosis of diseases of the alimentary system is made by giving 2½ ounces of pure barium sulphate (free from soluble barium salts for X-ray diagnosis) in 10 ozs. of butter milk and noting the progress of the meal by screening and skiagrams at various intervals. The medium being fluid takes the shape of the organ through which it passes and in this way pathological conditions of the oesophagus, stomach, duodenum, caecum, appendix, colon and rectal ampulla can be diagnosed.

Oesophagus :—The symptom usually present in affection of the oesophagus is dysphagia which may be caused by intrinsic or extrinsic causes. It is therefore always advisable to screen the chest before proceeding to investigate the condition of the oesophagus, in order to exclude aortic aneurysm and intrathoracic tumour. The condition of oesophagus is investigated by asking the patient to swallow a bolus of thin barium meal and noting its downward passage under the screen and subsequently taking a skiagram. In malignant strictures the barium is seen as a very narrow zig-zag line at the site of the stricture whereas a column of the meal is seen standing in a portion of the oesophagus above it (*Vide Fig. I*). In syphilitic strictures the picture is almost similar but the line of barium at the site of the stricture is not so ragged and irregular. In spasmodic strictures (cardiospasm) the spasm is seen to pass away after a time especially after the patient takes warm water or milk. Laryngeal cancer sometimes gives rise to reflex spasm of the oesophagus which may be mistaken for a stricture. Diverticulum of the oesophagus shows a pouch filled with barium along the line of the oesophagus and can be easily diagnosed with X-rays. Auricular dilatation sometimes gives symptoms of partial obstruction in oesophagus.

Stomach :—X-ray gives us a clear idea as to the (1) size (2) shape (3) position (4) tone (5) peristalsis and (6) emptying of the stomach. These points and tenderness to pressure at a suspected spot are determined under screen examination. Enlargements of the adjacent organs such as spleen, caudate lobe of the liver, gall-bladder, kidney, and the head of the pancreas give rise to rounded, smooth but persistent deformity in the outline of the stomach. Gastric ulcer is characterised by hypertonicity, hyperperistalsis and in typical cases by a *niche* in the outline of the lesser curvature with an *incisura* at the corresponding part of the greater curvature (*Vide Fig. II*). Rapid emptying occurs in early stages and stasis when there is cicatricial contraction. A bubble of gas is seen on the top of the niche when the ulcer is perforating. Pyloric ulcer is evidenced by persistent spasm at the pylorus, stasis, atonicity, dilatation and ptosis of the stomach. A post-pyloric ulcer is evidenced by hypertonicity, hyperperistalsis, spasm at the pylorus, irregular filling of the post-pyloric portion, rapid emptying in early stages and stasis in presence of cicatricial contraction. Duodenal ulcer is characterised by hypertonicity, hyperperistalsis, persistent deformity of the duodenal cap, tenderness to pressure over the duodenal cap and rapid emptying. Hyperperistalsis and rapid emptying of the

* Paper read at the Tenth All-India Medical Conference, 1933.

PLATE I

X'ray shows obstruction of the oesophagus due to cancerous growth.

PLATE II

X'ray shows gastric ulcer at the lesser curvature. Niche is seen filled with barium.

PLATE III

Cancer pyloric end of stomach
X'ray shows deformity at the pyloric end of stomach.

PLATE IV

Carcinomatosis of stomach. Note tube-like appearance of stomach, absence of peristalsis and rapid emptying.

PLATE V

X'ray shows calcified gall-stones.
T. B. affection of 2nd and 3rd lumbar vertebrae.

PLATE VI

Gall-Bladder—normal after the oral administration of cholepulvis.

PLATE VII

X'ray shows appendix adherent to the upper part of caecum after appendicitis

PLATE VIII

X'ray shows streak-like appearance of barium meal in colon suggesting chronic colitis.

stomach may be caused by inflammatory affection or neurosis of the stomach or reflexly by inflammatory condition of the adjacent organs such as duodenum, gall-bladder, appendix and colon. Adhesions of the stomach can be well diagnosed with X-rays. Tumours in connection with the stomach wall are evidenced by a persistent irregular deformity in the outline of the stomach. Malignant tumours are characterised by deformity in the outline and absence of peristalsis (*Vide Fig. III*). Fibromatosis of the stomach, commonly known as leather bottle stomach, is diagnosed by X-rays long before any suspicion of its existence can be clinically entertained. Every middle-aged patient who shows marked loss of weight with so-called *dyspepsia* should be immediately directed to undergo X-ray examination to exclude this condition. Fibromatosis of the stomach is characterised by very rapid emptying of the stomach which appears like a rigid tube with patent-pylorus and absence of peristalsis (*Vide Fig. IV*). Fibromatosis may be due to syphilis or carcinoma, and the clinical history with the Wassermann reaction helps in the diagnosis.

Gall-bladder :—Chronic cholecystitis usually causes adhesions of the pylorus and duodenum with the under surface of the liver and gall-bladder, so that it is evidenced on the film taken just after barium meal by (1) the pylorus being pulled over to the right side (hepatofixation) (2) persistent pylorospasm with hypertonicity and slightly rapid emptying and (3) a smooth sickle-shaped depression in the outline of the stomach near the pylorus. Gall-stones are visible as faceted ring-shaped shadows occurring in cluster when they contain sufficient amount of calcium. (*Vide Fig. V*). The gall-bladder can also be visualised by administering orally to the patient sodium-tetra-iodo-phenolphthalein and taking skiagram after 12 hours when the dye is secreted by the liver into the gall-bladder (*Vide Fig. VI*). This is known as Cholecystography. In very chronic inflammatory conditions of gall-bladder, the dye is not secreted into the bile and therefore the gall-bladder is not visualised. In such cases we have to rely on the findings of the stomach after the barium meal and the non-filling of the gall-bladder also can be taken as an evidence of cholecystitis.

Caecum and Appendix :—Barium meal reaches the caecum and appendix normally after 6 hours and so a skiagram is taken 6 hours and the next one 24 hours after the meal. Appendicitis is characterised by (1) stasis in the terminal coils of ileum (2) beaded appearance of appendix (3) residue in lower part of caecum and appendix even when ascending colon and terminal coils of ileum are empty and (4) tenderness to pressure over the appendix under screen examination. (*Vide Fig. VII*). It may reflexly cause hyperperistalsis of the stomach and spasm of the duodenal cap and therefore be mistaken for duodenal ulcer if subsequent skiagrams are not taken. Chronic inflammatory condition of caecum is also evidenced by (1) ileal stasis (2) irregular filling of the caecum (3) caecal stasis or sticking of the barium to the caecal mucous membrane and (4) caecal adhesions. Adhesions of the caecum and appendix are determined by taking skiagrams in lying down and erect positions and noting whether they move up in as the former position. as they do normally. Inflammatory and tubercular affections of the caecum and colon produce an almost similar X-ray picture and clinical history should be taken into consideration to distinguish between the two, though generally speaking adhesions of the caecum and rapid emptying of the colon are more in favour of tubercular affections.

Diseases of Colon :—Ptosis, displacement or any anatomical abnormality of the colon can be readily recognised in the skiagram. Chronic colitis is characterised by absence of haustrations so that the colon looks more or less like a uniform tube (*Vide Fig. VIII*). Idiopathic dilatation of the colon is characterised by marked stasis and great dilatation of the colon. Strictures of the colon are evidenced by marked stasis, dilatation of the proximal part and the presence of persistent narrow streak of barium at the site of the stricture. The margins of the streak of the stricture are smooth and regular in case of dysenteric or syphilitic strictures whereas they are irregular, interrupted and zig-zag in case of malignant strictures. The age and clinical history of the patient should also be taken into consideration, before making a diagnosis. Diverticulitis is evidenced by circular beads of barium seen outside the lumen of the colon. X-ray is useful in diagnosing adhesions of the colon. Barium enema is very useful in diagnosing obstruction in the colon.

Lastly, it is the duty of the radiologist to see that the radiological diagnosis he arrives at must explain all the clinical symptoms of the case.

X-ray Treatment: Its Scope and Technique *

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Bombay

It is with hesitation and diffidence that I approach this subject. I am afraid you may be disappointed as I have nothing new or original to offer. One must remember that it is given only to the very few to strike out of the common groove and work on original research. The average worker has to do his routine task, and the way in which that task is performed is an experience which, I think, is well worth recording before one's fellow-workers. I am giving you my impressions of the subject and the experience of practical working, the way I work it, and understand it, and I would very much like to know how you all feel about it. It is by an exchange of ideas alone that we can progress in any branch.

Rays impinging on the skin are partially absorbed and partially pass through. It is the rays that are absorbed and thus cause the desired effect and not those that pass through. Another important factor is the intervening healthy tissue through which the rays have to pass before reaching the diseased tissue. We have, therefore, to so regulate our working conditions that the diseased tissues receive sufficient quantities of radiations to produce the desired effect with minimum damage to the healthy tissues. This means selection of the proper rays, of the proper penetration to suit each particular disease. This is easier said than done but for all practical purposes we can classify diseases into three important groups (*i*) those requiring superficial treatment (*ii*) those requiring medium treatment (*iii*) those requiring deep treatment with maximum penetration.

Chronic skin lesions whatever their causation or pathology react remarkably to radiation. Particularly is this the case in *eczema* where a patch persisting for several years is seen to react after a few treatments. Contrary to general practice I prefer to use a higher K. V.—125—filtered through one millimetre of aluminium. The first dose is $\frac{3}{4}$ erythema followed up by smaller doses once a week. If efficiently radiated and a mild reaction kept up the patches do not reappear. If on the other hand, they do reappear owing to insufficient treatment or other etiological factors then their subsequent disappearance is a matter of difficulty. My reason for using a harder ray is that these patches extend deeper than they appear and moreover there is an additional margin of safety. Multiple patches of eczema are not very encouraging in their response. The very fact that they are multiple show a strong constitutional factor requiring correction.

Taenia affections of the scalp are not so common in this country. The results in these intractable forms are another triumph of X-ray treatment. Even if there is a solitary patch the whole head has to be treated otherwise it is apt to recur.

In *psoriasis*, results are not encouraging. It is pre-eminently a case for the internist.

Keloids, warts, callosities, furunculosis, chronic ulcers are some of the many conditions which though of minor pathological importance are benefitted by X-rays. Some of these conditions are very intractable and do not react to other lines of treatment. Take, for instance, a keloid. What can one do for such a condition ? Keloid reminds us of a very peculiar and characteristic effect of radiations, namely, that they will under certain conditions bring about resolutions of fibrous tissues. On the other hand, in other conditions they will stimulate tissue cells to form extra fibrous tissue and bring about fibrosis which plays such an important part in the treatment of various diseases.

The second great group of cases include *tuberculous glands, fibroids*, and *menorrhagias*. The results are most gratifying. The K. V. employed is 140 to 160, filtered through 4 mm. of Al or 0.2. mm. Cu.

It is now no longer necessary to submit the patient to a disfiguring operation. When glands are surgically removed they are apt to recur in other parts. Such cases form a large part of routine work. So many come with operation scars. The glands if softened are first aspirated and then radiated. If there are sinuses a preliminary course of ultra-violet exposures is given. The results are excellent. The same cannot be said of abdominal glands. These also react but unfortunately they succumb to pulmonary complications. Whether the radiations play a part in the sad ending it is

* Paper read at the Tenth All-India Medical Conference, 1933.

difficult to say. In any case such cases are undertaken with extreme care and some hesitation.

Fibroids and Menorrhagias yield with uniform success provided reasonable care is taken to exclude unsuitable cases. The usual contraindications are (1) septic condition of the tubes (2) soft and degenerated fibroids (3) very large fibroids causing considerable pressure (4) cases where malignancy is suspected. Even excluding such cases there is a large and comprehensive field. Regarding the question of age, there need be no hesitation when patients are over 35. But even younger patients may be treated provided other means have been tried and failed. Some of my patients have been young girls in whom there has been severe and most intractable hœmorrhages. When one is faced with a possibility of either giving radiation treatment or performing hysterectomy there ought to be no hesitation whatsoever. Obviously an artificial menopause is not desirable and aimed at in such a dose as will give breathing time and temporary cessation. This can be very satisfactorily accomplished by proper graduation of doses.

There is an impression among gynæcologists that if pregnancy supervenes the child is apt to be deformed and disfigured. Such an eventuality could perhaps take place if the patient conceives during the course of treatment. But apart from this there is no justification for such an alarm. Modern work on the subject does not support this.

The patient is benefited in every way. Fibroids shrink, fibrous adhesions are loosened, dysmenorrhœa is alleviated and the outlook of many nervous debilitated wrecks is considerably altered.

Regarding dosage there is no occasion for using intensive treatment. Semi-intensive divided doses give to my mind the best results. I generally give four successive daily doses through four portals. The treatment is stopped for a month and a second series is repeated after the menstrual period. Patients always have one menstrual period at least and in some cases slight irregularity persists for two or three months till finally menstruation stops altogether.

There is a distinct advantage in not rushing them through. More gradual the treatment, the milder the unpleasant menopausal symptoms, there is much less X-ray sickness and the patient can go about without the slightest disturbance.

Exophthalmic goiter and Hyperthyroidism are now referred more and more to the radiologist. Mild cases associated with palpitation, general nervousness, loss of weight, slight fever are fairly common and react readily. The serious cases associated with pronounced exophthalmos react but after several series of treatments. These are essentially cases for operation but it is just in these cases that serious risk is involved.

The essential pathological changes in this condition are such as lend themselves readily to radiation treatment. There is active proliferation of the cells lining the acina. Such cells are very vulnerable and are destroyed. Moreover, there is proliferation of connecting tissue producing fibrosis, further restricting the functional activity of the gland. In other words, the effects are similar to partial surgical removal. Surgery obviously would be an ideal procedure but for the serious risk. Moreover, many cases are inoperable owing to excessive enlargement or deeper involvement so that, we have to fall back on radiation therapy.

In the early days treatment had to be given almost daily for weeks but now our technique has considerably improved; exposures are fewer but more intensive and penetrating, improvement is always gradual and can only take place if the patient co-operates by taking complete rest in bed which is an important essential of treatment.

Spleno-Medullary Leukaemias are markedly benefited. The number of white cells drops, the huge spleen disappears and the symptoms improve. Unfortunately improvement is only temporary and sooner or later all the symptoms reappear and at last they reach a stage when they no longer react to to radiations.

Chronic Malarial Spleens hardly go down in size. There is much fibrous and connective tissue all round, that challenges any resolution.

We now come to the consideration of malignant diseases. Treatment of such cases is most depressing and disappointing not merely because of the intense suffering of our patients but more so as we feel that in spite of conspicuous progress in every way, better instruments, more precise technique, more intimate knowledge, we are still groping in the dark and are helpless in the majority of instances. Results are far from satisfactory. When the Erlangen Researches were first announced people thronged from all parts of the world. High expectations were held which unfortunately have not fructified. The technique for such cases is wildly divergent with different workers but there are some factors which have received unanimous approval. There are no two views about using the maximum voltage, heavy filtration, maximum distance, several portals of entry and a lethal dose directed on the growth. The average working conditions are 200 K. V., 0.5 Cu. 50 to 75 centimetres distance.

An experimental plant is working at New York which is activated at 900 K. V. The radia-

tions are expected to be of the hardness of the *gama* rays of radium. Question arises "Is there any advantage in using greater kilovoltage than at present ?" The whole art and essence of treatment is to deliver a damaging dose to the malignant cells with the least possible damage to the healthy cells. Other factors being same this is best achieved by using higher and higher voltages. The higher the voltage, the greater is the proportionate damage to the malignant cells as compared to the heatlhy cells. If, for instance, using 200 K.V. the ratio of damages is 1 : 2, then using, say, 900 K.V. the ratio is expected to be altered say 1 : 5 . The practical outcome of all this is yet to be seen.

Regarding dosage determination all are agreed. The fundamental basis is the biological effects produced, the *Erythema dose* still holds ground. But whereas few years ago its determination was a matter of personal observation and experience, now-a-days with the advent of reliable *Ionometers* this can be done with precision. In other words dosage has been standardised, 800 R. is now recognised as being equivalent to an erythema dose. This is an enormous advance.

The next important step is the selection of portals. Hoeff Elder uses and has devised the three portals method, each portal at an angle of 120°. The Erlangen and the French School believe in multiple portals. Elaborate charts are available but, I believe, common sense and a clear idea of the anatomy helps to solve this important question more than anything else.

Regarding the giving of the actual dose there is serious divergence of opinion. The German School, I believe, still persists in giving the whole dose at one sitting. The French School on the other hand splits the dose in anything from eight to sixteen sittings. There appears to be a distinct advantage in the French method. Neoplastic cells are in various different stages of development and division at any one moment. In certain stages the cells are radio-sensitive ; in other stages they are radio-resistent. By hammering at the growth constantly in divided doses we are able to catch the cells in their radio-sensitive stage. The same idea is underlying the recent modification in the application of radium needles. Needles are now used in a much smaller strength and for a longer time. Moreover when the doses are divided a much greater quantity of radiation can be given.

Breast cases demand our first attention. Early operation with prophylactic radiation offer the best chances of cure. There is a school of workers who would use radiation even in operable cases. In doing so, I think, the Radiologist is taking an enormous risk. After all what can be removed by a clean and radical operation cannot possibly be achieved by radiation treatment. Prophylactic treatment after operation has reduced the chances of recurrence considerably. One thorough efficient prophylactic course is to be aimed at. There is no point in repeating it at frequent intervals. Such is the considered opinion of an eminent authority like Prof. Wintz.

In treating recurrent growths of the breast damages to the underlying lung tissues have got to be borne in mind and prevented. This can be done by so grouping the portals and the rays in an oblique manner that while including the growth only the periphery of the lungs is touched.

In two important regions radiation therapy has displaced surgery. One is the *tongue* and the other is the *cervix*. Radium needles are embedded in and around the tongue and are implanted in the cervix and deep therapy is given to the neck and the pelvis to deal with the glandular area. In early cases the results are very satisfactory.

Results in inoperable cases can only be disappointing from the point of view of complete cure, and relief from symptoms is all we can expect to give. Pain diminishes, ulcers heal, patient is made more comfortable and in any case has the satisfaction of feeling that something has been done for him. After all mental factors and processes play a large part in human sufferings.

A large measure of success is achieved in *epithelioma* and *rodent ulcer*. Such cases can be kept under satisfactory check by intensive radiation.

A great deal has been done in recent years but much has yet to be achieved. Constitutional conditions and factors which encourage the production of the tumour have yet to be investigated and studied and unless we know what they actually are and are able to deal with them local treatment alone, as at present, must play a minor part in bringing about the cure of the patient.

Preparation & Standardisation of Anti-gasgangrene Serum & its Application in Human Medicine*

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The Medical Research Committee upon Anærobic Bacilli and Infections are of opinion that "Acute gasgangrene may result from at least three types of pathogenic anærobic bacilli; these in their order of frequency are (1) *B. Welchii* (2) *Vibrion septique* and (3) *B. Oedematiens.*

"In addition to these, many other anærobes may be present in wounds. The exact part which they play in the production of Gasgangrene is not clear. In pure culture, they cannot be regarded as pathogenic for laboratory animals" (*Special Report Series No. 39*).

As *B. Welchii* has been found to be the most important organism, so far as the application of the antitoxin in human medicine is concerned, this organism only has been taken up in details for the present purpose.

B. Welchii, was described first by WELCH and NUTTALL, as *Aerogenes capsulatus* in 1892 and within a few months, the same organism was obtained by FRÆNKEL, who dealt with it as the *Gas Phlegmon Bacillus* (*Fraenkel's Bacillus* of German Literature) and it was named once more in 1898 as *B. Perfringens* by VEILLON and ZUBER (of the French).

SIMONDS divided the *B. Welchii* into 4 groups:

Group	I.	Ferments inulin-& glycerine.
,,	II.	,, glycerine, not inulin.
,,	III.	,, inulin, not glycerine.
,,	IV.	Does not ferment glycerine or inulin.

Different strains of Gas Bacillus were subsequently classified by HENRY, who recognised 4 types. Types II and III correspond exactly to SIMONDS' Groups II & III. Type I of HENRY corresponds to Group IV of SIMONDS and type IV of HENRY corresponds to Group I of SIMONDS so far as their effects upon inulin and glycerine are concerned. Being essentially a saccharolytic organism all these types and groups have the same characteristics of fermentation of glucose, levulose, galactose etc. This organism produces practically no proteolysis.

PREPARATION OF WELCHII TOXIN

After working with several media, we have been able to get good toxin with the cooked meat medium which consists of beef infusion broth and fresh sterile muscle tissue at the bottom of the flask in the proportion of 2 handfuls of cooked meat in 2-litre of broth. The hydrogenions of the medium are adjusted to the optimum degree of concentration which is pH-7·2. The flasks containing media, are boiled thoroughly for 30 minutes, to expel as much of the dissolved oxygen as possible and then rapidly cooled to about 40°C. A layer of sterile vaseline is applied on to the top of the liquid and inoculations are then made quickly. We have taken advantage of the work of KAHN, as reported in the year 1922, for producing perfect *anærobiosis*, by rejecting the use of pyrogallic acid and sodium hydroxide, vacuum exhaustion, hydrogen displacement etc. which have been found unreliable by him.

Flasks are inoculated after heating them for ½ an hour in KOCH's steriliser and rapidly cooled to 40°C., with 15 c. c. of a 24 hours' culture of *B. Welchii*, obtained from abroad.

We are not yet convinced about the necessity of passing the strain through 2 or 3 pigeons before using in the inoculation of the flasks as has been advocated by BENGSTON. The flasks are incubated for 21 to 24 hours and filtered. The toxins formed, are controlled and if found sterile, both ærobically as well as anærobically, are tested for M. L. D. In controlling anærobically, a special point is worth

* Paper read at the Tenth All-India Medical Conference, 1933.

mentioning. At least 100 c. c. of the toxin from each lot is cultured in cooked meat medium and incubated for two days. Even if one spore is present in a flask of toxin to be utilised for the process of immunisation, it is to be detected ; otherwise disastrous results may follow, as we found in the case of two of our horses described below. Those lots where more than 2½ c.c. are required as test dose for a pigeon of 350G. are rejected and not utilised for immunisation of horses. Since pigeons had proved to be highly susceptible to infections of *Welchii* and to correspond with the characteristic pathological reactions, HERTER at FLEXNER'S suggestion employed this animal and ever since 1917, pigeons have been taken as the standard animal for this work.

We have found it not advisable to add glucose in the toxin production, though advocated practically by all the workers, in this line, as the *B. Welchii* has been found to expend its energies on the fermentation of glucose present at the expense of toxin formation, particularly if the organism is grown in an alkaline medium.

We have been encountering no difficulties in getting good amount of *Welchii* toxin with ¾ c.c. as M. L. D., for pigeons (350 G.), ¼ c. c. for mice (18 to 20 G.) and 2 c. c. for guinea pigs (250 G.). This we consider as reasonable titre for the purpose of immunisation of horses.

The nature of gases produced during the cultivation of *Welchii* had been studied previously by other workers and CO_2 and H_2 gases have been identified. We are working on this line in collaboration with the UNIVERSITY COLLEGE OF SCIENCE at Calcutta and our findings will be reported later on.

PRODUCTION OF ANTI-GASGANGRENE SERUM

For the manufacture of *Welchii* anti-toxin, we have used horses which have been previously injected with a course of tetanus toxin. With regard to the choice of horses, and the method of immunisation, we have been able to produce good serum with country-bred horses as well as with Australian and the usual method followed for immunisation against diphtheria has been found to be successful. We have not used '*anaculture*,' but toxins and toxoids (·2% formalinised), both giving equally good results. We had the misfortune of losing two of our good *Welchii* anti-toxin producing horses. After being fully immunised they had been bled for 2 or 3 times. They did not show any signs of fever, nor were there any manifestations of any toxic effect at the time of the last injection. Twenty-four hours after the injection of the last dose of toxin the horses were found sick, getting twitchings all over the body without having lock-jaw or stiff-neck. Peritonitis set in. The abdomen was found full of gas as if it was going to burst open. Eyes became injected and they were found restless to an extreme degree. The poor animals died within 24 hours retaining full consciousness to the last, inspite of the injection of huge doses of P. D. Serum intravenously, and subcutaneously. The restlessness exhibited was of a peculiar type, the animals trying to jump out of the enclosure even towards the end.

METHOD OF STANDARDISATION

BULL supplied a serum which bore a label indicating the unitage. He used as his unit the quantity of serum, which neutralised an amount of toxin, equal to one lethal dose for the pigeons. BULL'S unit was found to correspond with the unit adopted by BURROUGHS WELLCOME LABORATORY, and accepted also by MEDICAL RESEARCH COMMITTEE who fixed up as unit, twice the amount necessary to neutralise twice the lethal dose for mouse.

But recently, a standard has been fixed by U. S. Public Health Service. *Anti-Welchii* Serum is sent out from there, mentioning units on the label, as the standard serum. 1-100 unit of standard serum is mixed with varying amounts of toxin to determine the smallest dose of toxin, which will overcome this amount of serum and kill the pigeon of 350G. within 24 hours. This dose of toxin is known as the test dose. This test dose of toxin is then mixed with varying amounts of serum to be tested and is then injected with a second series of pigeons, after incubating the toxin anti-toxin mixtures for 45 minutes at 37°C. That amount of serum which gives protection for 24 hours against the test dose of the toxin shall be considered to contain 1-100 unit. The dose of toxin as well as the anti-toxin must be proportional to the weight of the pigeons, the standard weight being 350 G., the testing should be done on pigeons weighing between 325 to 375G.

As is the case with tetanus and diphtheria serum, an excess of 25% is allowed to provide for deterioration of serum.

We arranged the following set of experiments and repeated it for six successive times, with virtually the same results as noted below :—

Cage No.	Pigeon marked.	Weight of the pigeon in G.	Unit of serum injected per 350 G.	Actual unit injected.	Dilution of serum.	Actual quantity of the diluted serum in c.c.	Time of injection of serum.	Toxin lot.	Quantity of toxin injected per 350G. in c.c.	Actual dose of toxin injected in c.c.	Time of injection of toxin.	Time of death.	Hours survived.
													H. M.
1	Red ...	360	·01	·0103	1·99	1·03	10 A.M.	23W	2 c.c.	2·06	10 A.M.	2-45 P.M.	4-45
	Black ...	370	,,	·0106	,,	1·06	,,	,,	,,	2·11	,,	3-15 P.M.	5-15
2	Red ...	375	,,	·0107	,,	1·07	,,	,,	,,	2·14	11 A.M.	5 A.M.	17 hrs.
	Black ...	345	,,	·0099	,,	·99	,,	,,	,,	1·97	,,	4 A.M.	16 hrs.
3	Red ...	350	,,	·01	,,	1	,,	,,	,,	2	12 Noon	survived	
	Black ...	360	,,	·0103	,,	1·03	,,	,,	,,	2·06	,,	6 P.M. next day.	30 hrs.
4	Red ...	330	,,	·0094	,,	·94	,,	,,	,,	1·89	1 P.M.	survived	
	Black ...	340	,,	·0097	,,	·97	,,	,,	,,	1·94	,,	,,	
5	Red ...	360	,,	·0103	,,	1·03	11 A.M.	,,	,,	2·06	10 A.M.	3 P.M.	5 hrs.
	Black ...	350	,,	·01	,,	1	,,	,,	,,	2	,,	3-30 P.M.	5-30
6	Red ...	370	,,	·0106	,,	1·06	12 Noon	,,	,,	2·11	,,	2-30 P.M.	4 hrs. 30 m.
	Black ...	375	,,	·0107	,,	1·07	,,	,,	,,	2·14	,,	3 P.M.	5 hrs.
7	Red ...	345	,,	·0099	,,	·99	1 P.M.	,,	,,	1·97	,,	2 P.M.	4 hrs.
	Black ...	360	,,	·0103	,,	1·03	,,	,,	,,	2·06	,,	2-15 P.M.	4-15
8	Red ...	360	·015	·0154	,,	1·54	11-30 A.M.	,,	,,	2·06	,,	9 P.M.	11 hrs.
	Black ...	350	·015	·015	,,	1·5	1-30 P.M.	,,	,,	2	,,	7 P.M.	9 hrs.
9	Red ...	350	·02	·02	,,	2	1 P.M.	,,	,,	2	,,	12 Noon next day.	26 hrs.
	Black ...	345	·02	·0198	,,	1·98	,,	,,	,,	1·97	12 Noon	10 P.M. next day.	34 hrs.

Prophylactic and Curative Value

Though Bull & Pritchet had shown by experiment on laboratory animals the prophylactic and curative value of *Anti-Welchii Serum*, we failed to get the same curative results and in our experience with our own serum, that of P. D. & Co., and also of the standard serum supplied by the U. S. Public Health Service, the curative value of the serum is practically *nil*, if not injected early in the infection.

It will be noted that the dose of serum injected, was even increased in the last two series to get curative effect if possible in bigger doses. The result will show the prophylactic value of the *Anti-Welchii Serum* to a remarkable extent and will demonstrate the inefficacy of the serum in curative effect, excepting when injected early.

Indications

B. Welchii finds a normal habitat in the human intestine, as well as in the intestines of pigeons etc. It is only under abnormal conditions depending upon the reaction of the medium, where it is present, that toxic condition is found. In all cases, of peritonitis or of intestinal obstruction or appendicitis there is every chance of this bacillus developing pathogenic properties. This also finds entry into abnormal places like uterus, through the intestines and in some cases of puerperal septicaemia, *B. Welchii* plays an important role.

In all cases of abdominal operations either surgical or gynæcological involving the intestines in particular *Welchii anti-toxin* should be injected as a prophylactic dose, as freely as Anti-tetanic serum is used in cases of street wounds.

Welchii anti-toxin will be found beneficial only if injected early in the disease, and more particularly to prevent infections during street wounds and dirty clothes, from where this organism has frequently been isolated.

In conclusion, I acknowledge with thanks, valuable co-operation and assistance from Drs. J. Naha and Satish Ch. Bhattacharjee of our laboratory during this work.

Studies on the Periodicity of Defective Function of Human System as Distributed in the Phasial Cycle of the Moon

Part IV

H. CHOWDHURY, M.SC., M.B.

Rangoon

DISCUSSION

In the above series of experiments an attempt to find out a periodicity of defective body function expressed in terms of lunar cycle has been made.

In the first group of experiments where *provocative polyuria test* have been done we see a clear indication of defective kidney function as shown by diminished elimination on certain days as distributed in phasial cycle of the moon. Curiously enough this has a good coincidence with the old statements of the Hindu Sages who ascribed these days as being days when health is ill-disposed. The days are the 3rd, 9th, 13th and 14th day following either the New or the Full Moon days, including the New and the Full Moon days themselves. Question at once comes to one's mind as regards the influence of Relative Humidity on the evaporation from the skin. Consequently Relative Humidity was also taken into account for a period of one year. The mean curve here also shows an indication of the possibility of greater evaporation from the skin round about these days. Recently, M. W. GOLDBLATT from London has shown that the total water exchange of the body bears no relation to the loss of water through the skin and that diminished extra-renal excretion was associated with cases of oedema and polyuria. So in my cases also, I do not know how much this Relative Humidity has influenced the elimination of water through the skin. Even if it has done so it is worth while noting such events as they come off fairly regularly in a cyclic fashion. So far is about the mean curve in either events but if individual curves are considered we see a marked shifting of these days backward or forward as distributed in the phasial cycle of the moon.

In trying to explain it, the influence of the Moon, the nearest of all to the Earth, on the tide was considered. To make an accurate study I plotted the curve of the heights of water level in one of the local rivers (Rangoon river) as determined on the various days in the phases of the Moon for a period of one year. Here the highest and the lowest level of water heights vary within a range of 3 to 4 days from month to month (as shown). So it is not unlikely, that such shifting of the days of defective elimination should occur in the case of man also, when individual curves for different months are considered.

It has been said that the lives on Earth came out of the Sea. So it is only possible that the system should yet have things similar in structure and function to those of the water of the sea.

KAPILLA, the father of evolution theory of the Vedic age, whose views are same as those of HERBERT SPENCER of later years, in his *Sankya* system said "Laws of Nature are uniform and regular throughout."

MANU, the greatest of all social and religious reformers of the Hindus and of the Vedic age also mentioned in his *Manu Samhita* as regards the birth of the Universe :—

सोऽभिध्याय शरीरात् स्वात् सिसृक्षुर्विविधाः प्रजाः ।
अपएव ससर्ज्जादौ तासु वीजमवासृजत् ॥—मनुः ।

By which he meant that :—

In the beginning was the *Will* and the *Will* was followed by the birth of *Water* and it is from this *Water* that the Universe with all its diverse manifestations came into being.

यावन्तोहि भावविशेषाः पुरुषे तावन्तोहि लोके ।—आयूर्व्वेद ।

That the human body possesses everything that exists in the Universe.

Views on the similarity of blood plasma to the sea water had been confirmed by SIDNEY RINGER'S extensive work on isolated heart in 1880.

Investigations of similar nature were also carried out by BUNGES in 1902 on land vertebrates, by MACCULLUM in 1903 on comparative study of plasma and sea water and also by NENKIRCH on rabbits' intestines. In all these experiments it has been shown, beyond reasonable doubt, the in-

timate relationship between the sea water and the blood plasma.

A careful examination of the tide curve shows that on the 2nd, 3rd and the 4th days following the New and the Full Moon days a great amount of water is drawn towards the land by rivers from the sea, on the other hand comparatively little amount are attracted towards land on other days specially on or about the 10th days following these days when most of the water remain in the sea.

Here one can hardly refrain from wondering a little in the field of speculation. If the blood vascular system is compared to the sea and the lymphatic and tissues spaces as the tributaries of the same, we can have a somewhat comprehensive idea of the observed facts. But difficulty arises again about the sun and the moon substances in the body. CHARAK and SUSRUTA and almost all the Hindu physicians of the Vedic age compared *pitta* humour as the product of the Sun substance and the *kauf* those of the Moon substance in the body. As regards the sites of their origin, they said :—

ऊर्द्ध नाभेरधस्तालोरुर्द्ध भानुरधः शशी ।—हठयोग

Which literally means :—

That the Moon element exists over the palate and the Sun element over the naval region.

It may be that before they are actually formed in the sites of formation they circulate in the blood in the same way as the elements of milk do before it is actually formed and secreted by the mammary gland cells.

CHARAK and SUSRUTA have also mentioned that as the sun during the rainy season cannot exert its full influence on the earth due to the accumulation of clouds in the sky so also in the body the sun elements are resisted from exerting their influnce as digestive juices due to the accumulation of *kauf* substances. So it is not unlikely that this colloidal *kauf* substance may accumulate in the cells and intercullar substance thus attracting salts and water. Thus the transfer of the water to the tissue can be explained to some extent.

Such remarks can be substantiated by the experiment No. 5 where simultaneous readings of urine volume, corpuscular volume and blood pressure have been taken, both immediately before and one hour after, the administration of 500 c.c. of water. Here the proportionate diminution of plasma volume, with fall of blood pressure and diminished urine flow have been observed. Urea concentration test with 15 G. of urea with 100 c. c. of water with simultaneous study of blood urea was also made. Here also we see a corroborative evidence of defective kidney function (as expressed in terms of urea % in urine) with low blood urea—which is possible only if urea and water are retained in the tissues.

Determination of fasting blood sugar and nitrogen partition of urine were also undertaken to verify the statement prohibiting taking of rice, meat, etc., on the 8th, 10th, 11th, 14th, and 15th waxing and waning days. The curves show a clear evidence of defective function of liver on these days.

The low fasting blood sugar can be explained by slow absorption associated with increased retention in the tissues, which becomes much more evident by administration of glucose with water (Experiment 9).

One can also easily surmise from the statements of the Sages of the Vedic age that on these days the cells of the body in general must be working at great disadvantage. So not only the absorption but also excretion suffer from such unnatural state.

To verify these assumptions further tests on glucose tolerance were done which conclusively proved the defective function of the liver and the tissues as regards the utilisation of the same on these days (Experiment 8).

The next experiments were carried on an oedema patient. It was found that on these days associating with defective function of the cells as evidenced by death, etc., there were increased retention of sugar in the tissues (Experiment 10).

W. STARK in 1769 and F. G BENEDICT and R. D. MILNER in 1907 showed retention of water with carbohydrate diet. JAMBLE, ROSS, TISDOLL and HAELZEL also mentioned the loss of extra cellular water due to fasting. BRIDGES could not find by experiment on liver retention of 3G. of water by 1G. of glycogen.

MCCLENDON suggested that with glucose the e is redistribution of water in the body. He experimentally showed transference of water from muscles to the skin in a mouse (due to carbohydrate diet).

It has always been the custom with the old *Ayurvedic* physician not to give any rice diet to a patient suffering from oedema because rice increases the oedema.

Here also probably the same phenomenon occurs, *viz.*, greater retention of sugar in the tissues drawing more fluid from the blood.

The observation of ROWNTREE and GERAHAGHTY, that the excretion of water does not follow *pari passu* with the excretion of drugs ; so that with smaller doses there may be increased

output whereas with bigger doses there may be the opposite results—can be well explained in the light of the periodicity of defective function. Similarly ABBARRAN's findings that :—

1. the healthy kidney was most ununiform in its function,
2. the diseased one was more uniform,
3. and sometimes with large quantity of water no polyuria could be had,

can be explained.

WIDAL observed raising of blood pressure in those cases where elimination by the kidney was defective. He has also mentioned that retention of water in the tissues was possible but always with retention of chlorides and urea.

PRIESTLY was of opinion that water drunk to the extent of 5 litres could be eliminated by the kidney without any alteration of blood crystalloids and the hæmoglobin content in the course of 4 to 5 hours. He also suggested that the extra water was accommodated into the tissue spaces, muscles and skin, with some crystalloid of the blood for the up-keep of the osmotic tension for the time being. The maximum rate of elimination of water per hour by the kidneys according to him was 1300 c.c. which could not be maintained for a long time. But according to him an elimination of 750 c. c. per hour could be maintained for a long time.

My experiments as far as the transfer of water and crystalloid to the tissues are concerned, bear evidence to his experiments. But I cannot reason out how such a thing was possible without marked alteration of blood crystalloids, neither can I see my way to take it for granted that the kidneys will act, if healthy, uniformly every day in the way suggested.

Observations with mineral water have also been made by VACQUEZ, ALBERN, MERKLEN and others where they found greater elimination during the daytime by the healthy kidney. Spa practitioners concurred with the same view. GUBB observed that water was not always eliminated when drunk to flush the system. It raised the blood pressure to an uncomfortable extent. In some it was eliminated very soon, in others very late.

From these findings we can see that similar defects in water elimination was also seen by others but explanations were wanting as obviously solitary experiments were done on indefinite days, it might have something to do with the periodicity as found in terms of the phasial cycle of the Moon.

The uniformity of diseased kidney and non-uniformity of the healthy kidney in eliminative function as observed by ALBERN can be similarly explained, as in diseased kidney the fluctuation towards better elimination on certain days in the phasial cycle of the moon is not possible,—all the cells being uniformly unhealthy.

The last set of experiments on skin resistances by electrocardiograph shows deflections of the fibre to vary a good deal either to the left or right of the isoelectric point (the zero of the scale) when a current of 1 *milli-volt* is allowed to pass, the sensitivity of the fibre being adjusted to 10 m.m. deflection before the leads were used.

When making such observations one cannot refrain from thinking of internal secretion specially adrenalin which has the most effect of constricting blood vessels of the skin and the intestines.

DENSHAM suggested the possibility of the deformation of the superficial layer of epidermis by the vasoconstriction of the skin vessels which diminishes the electrical resistance of the skin to a great extent. Similar diminution of electrical resistances during mental effort due to vasoconstriction of skin, raising the blood pressure was described by him as " Psychogalvanic reflex."

Very recently KUGALMANN has made some observations on the relation of insulin to adrenalin where he injected 30 units of insulin to healthy men and saw after 30 minutes the fall of blood sugar, when it came down to ·06% the blood pressure went up. But the blood pressure went down on injection of glucose in such cases when the blood pressure went up simultaneously. But if glucose was injected with insulin no such thing occurred.

From these it may be surmised that in all probability the adrenal secretion falls below normal on such days. So the insulin works unhampered ; low blood pressure, diminishing the urine secretion (as to the raised blood pressure, the blood sugar ought to go down to ·06 %).

Before concluding, I think it is worth while considering the experiments done by TITUS and his co-workers on blood pressure of eclampsia cases where he suggested the hypoglycæmia to be the causative factor of eclampsia.

In addition LAFERTY brought forward also the pathological findings of the various organs, like liver, etc., which were found depleted of all carbohydrate and glycogen. Similarly, he found the internal secreting glands like suprarenal, pituitary, thyroid and pancreas, *i.e.*, those concerned with carbohydrate metabolism altered in structures to a great extent in this disease.

Similar pathological changes of fœtal and maternal organs were also observed by me in 1925 when the interpretation of these were difficult.

But at the present moment if one compares the cases of eclampsia as distributed in the phasial cycle of the Moon with that of similar curves of blood sugar of healthy men fasting for 14 hours, it is observed that on such days, even in health, the blood sugar goes down in man. So it is quite possible that such a change will be more marked, in a woman near about the time of parturition, when she is carrying out the metabolism for herself and her child.

SLANDER and HARRISON (of Johns Hopkin's Hospital) to combat the hyperglycæmia in eclampsia treated his patients with insulin. But unfortunately he never repeated TITUS's experiments.

CERI CERI suggested the mobilisation of glycogen to lactic acid from muscle by adrenalin during excessive muscular exercise and also in oxygen deficiency. This lactic acid was converted into glucose in the liver. So the conclusion arrived at by LAFERTY was as follows :—

That either one or all of the following factors may be involved in the disease :—

(*a*) continuous heavy drainage of glucose from maternal blood to fœtus,

(*b*) excessive consumption by the maternal tissue,

(*c*) increased insulin in maternal blood,

(*d*) an inadequate supply of glucose due to faulty alimentation.

Under such conditions when the store was exhausted liver starts, supplying glucose from protein ; when that fails eclampsia comes in. The fits were supposed to release lactic acid from the glycogen of muscle to supply materials which liver could convert into glucose.

CONCLUSIONS

1. That on the third, ninth, tenth, thirteenth and fifteenth of waxing or waning days the tissue cells in general are less efficient in their functions.

2. That associated with these there are retention of colloids, crystalloids and water in the tissues.

3. That it is probably due to these changes in the system that one's appetite, muscular and mental activities are less than par on those days.

4. That it may be profitable as a preventive medicine if these days as expressed in terms of the phasial cycle of the Moon are treated as periods when the system works at a disadvantage.

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A Note on the Correlation between Chemical Structure and Pharmacological Action of Embelin

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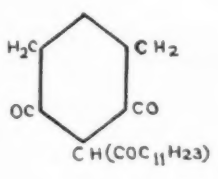
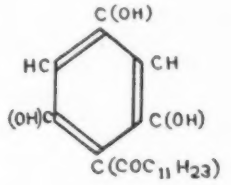
The structure assigned to a compound must be such that it should be a complete expression of its behaviour, not only chemical and physical but also pharmacological. Many attempts have been made to correlate chemical structure and pharmacological action of anthelmintics. HALL and WIDGOR (1917-18) made tentative efforts at such a correlation. More complete work was later (1909-23) done by CAIUS and MHASKAR [1], particularly in connection with the remedies of hookworm disease, but they were unable to find a common denominator of the dissimilar drugs found effective, and they did not continue in an attempt to build a series of related drugs and trace anthelmintic correlation. They pointed out the cumulative effects of the halogen atoms and the free phenolic hydroxyl group, also that of the peroxide structure in ascaridol. The importance of the lactone group has been traced by TRENDELENBURG[2] but has since then been denied by some though recently reaffirmed by OETTINGEN [3]. An attempt to group common anthelmintics in definite classes according to similarity in chemical structure and analogous pharmacological action has also been made by GREEN[4] (1931). He has suggested the following six groups: (1) the santonin or lactone group, effective especially against roundworms; (2) halogen group or carbon tetrachloride group effective against hook-worms; (3) thymol group also particularly effective against hook-worms; (4) group of petroleum ether, naphthalene, naphthol, etc.; (5) group of phloroglucinol derivatives, effective against tapeworms; and (6) the group of alkaloids like arecoline and pelletierine.

NOW PARANJAPE and GOKHALE[5] have shown that *embelin* is effective against tapeworms but has no action on hookworms, roundworms and whipworms. It would, therefore, be interesting to find out in which of the six groups suggested by GREEN *embelin* could be placed and what group or configuration is responsible for this specific action against tapeworms. *Embelin* has no lactone group, nor has it a naphthalene structure. If it resembles in structure any of the groups suggested by GREEN it is the phloroglucinol group, *if* the formula for embelin tentatively proposed by KAUL, RAY and DUTT[6] be accepted which shows it to be a lauric acid derivative of phloroglucinol, thus



Keto-form.



Enol-form.

If *embelin* is a derivative of phloroglucinol then it fits in with the other anthelmintics which are specific against tapeworms and which are also derivatives of phloroglucinol, *viz.*, (1) filicic acid and aspidinol from the rhizome of male fern, Aspidium felix mas (suartz), (2) Rottlerin from Kamala resin, and (3) Kosotoxin and Kosin from Kosin resin. Now HASAN and STEDMAN'S[7] formula does not show it to be a derivative of phloroglucinol but their formula is correct as it appears to be from their synthesis of embelin and from the fact that the mixed natural and synthetic product does not show any depression in the melting point. It may, however, be noted that HASAN and STEDMAN'S formula

(*a*) does not account for the crystalline condensation products which embelin is found to give, according to KAUL, RAY and DUTT with two molecules of aldehydes and two molecules of nitroso compounds showing the presence of two reactive methylene groups;

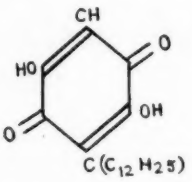
(*b*) does not account for the formation of a tetraoxime by embelin (KAUL, RAY and DUTT);

(*c*) does not explain the tautomeric nature of *embelin*.

These facts show that HASAN and STEDMAN'S formula is not a complete expression of the behaviour of embelin and does not give an entirely satisfactory explanation of its many properties; *e.g.*, chemical as shown by KAUL, RAY and DUTT and pharmacological as shown by PARANJAPE and GOKHALE if GREEN'S classification given above be accepted. But if one is inclined to accept HASAN and STEDMAN'S formula as it appears to be correct from its synthesis then it would be necessary to extend GREEN'S proposed classification correlating structure and anthelmintic action so as to include also the following group, *viz.*, derivatives of quinone and hydroxy quinone group. It would also be necessary in order to elucidate this question further to carry on the following investigations, *viz.*

(1) to isolate anthelmintics like *embelin*, specific against tapeworms from other natural drugs;

(2) to prepare compounds analogous and related to the synthetic product prepared by HASAN and STEDMAN, *e.g.*;



(3) to prepare compounds of phloroglucinol with different organic acids agreeing with the formula proposed by KAUL, RAY and DUTT for *embelin*;

(4) to prepare compounds analogous and related to all Aspidium and if possible to filicic acid containing more than one phloroglucinol nucleus;

(5) then to study the pharmacological action of all these substances both natural and synthetic and finally to compare their results.

Compounds of the types mentioned in (2), (3) and (4) above are under preparation.

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A modified Wu's method for Micro-estimation of Haemoglobin and the Haemoglobin content of Indian blood as determined by this method

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Introduction

The methods for determination of hæmoglobin in blood comes roughly under three groups :—

(1) *Colorimetric methods* :

(*a*) Blotting paper comparisons which give very rough idea.

(*b*) Haldane-Gower's hæmoglobinometer which gives rather low results and in which the standard solution does not keep well specially in tropical climates.

(*c*) Sahli's types either provided with standard solution or prisms—the latter being preferable—gives better results than the preceding but the results are higher.

(*d*) Newcomer's.—This instrument is the best as the standard glass may be compared in a colorimeter with the treated blood. But the apparatus is costly.

(2) *Chemical methods :* The best known of which is Wong's[6] or Reis-chakmakjian method[8] which depend on iron estimation of blood.

(3) Gasometric method such as O_2 capacity determination. This method gives the most correct result but requires expert handling of special apparatus.

But all the above methods suffer from one serious drawback in that the more accurate of them require 20 c. m.m. or more of blood which may not be obtained freely flowing and in severe cases of anæmia where repeated examinations may be necessary, such methods may be impractical or harmful.

In 1923, Wu first employed the pseudo-peroxidase re-action of blood with Benzidine for estimation of blood[11] and lately Bing and Baker developed the method into a microestimation by which hæmoglobin could be estimated accurately in very small quantities of blood.[1] The present work was undertaken on the line of Bing and Baker's modification of Wu's method. The difficulty and the inaccuracy involved in exactly measuring such small quantities of blood as ·005 c. c. is far outweighed by two factors : (I) accuracy of the method, which though a colorimetric one never gave an error of more than 5%, (II) small quantities of blood can be had freely flowing and need not be milked out and thus one of the principal sources of error in Biochemistry of blood *i. e.*, contamination of the blood with tissue extracts, is obviated. Another advantage of the method is that it is a very simple one and does not require any reagents nor special apparatus except an ordinary colorimeter which is now a standard equipment in all biochemical laboratories.

The method may also be employed for qualitative determination of minute quantities of hæmoglobin in urine, stool or plasma (after hæmolysis). It is a distinct improvement upon the old technique of Benzidine method of detection of "occult" blood and is extremely delicate. 1-1,000,000 dilution of blood gives a distinct colour change. The colour intensity being dependent on the amount of hæmoglobin, the amounts of this in stool, urine or plasma can be roughly assuaged in this way. A positive reaction is the immediate blue or green colour which later on changes to red brown colour, not very much unlike old Benzidine solution itself.

Principle of the Method

The well-known Benzidine test for occult blood is employed, the colour changes in the test being dependent on the pseudoperoxidase reaction of blood. On addition of Benzidine solution and H_2O_2 to traces of hæmoglobin solution there is an immediate change of colour to blue or green and this colour slowly changes to a dark brown colour and is intensified. This colour, on addition of acetic acid, becomes transparent and changes to sepia. The intensity of these colour changes being dependent on the amount of hæmoglobin, an unknown solution of hæmoglobin is treated with these reagents and the colour developed is matched

against a standard solution of hæmoglobin, similarly and simultaneously treated and from this the hæmoglobin value of the unknown is calculated.

Reagents required

(1) 2% *Benzidine solution.*—Dissolve 2G. of good quality Benzidine (we find Merck's quite satisfactory if freshly opened) in 20 c.c. glacial acetic acid in a bath at 50°C. When dissolved take out from bath and allow the mixture to cool. Dilute to 100 c.c. with distilled water. Decolourise by shaking with Merck's medicinal charcoal (about ·5 to 1 G.) and allow to stand for 15 minutes with occasional stirring. Filter and store in dark in brown bottle. If the solution colours on keeping, decolourise again before use[2].

(2) 10% *Acetic acid solution.* Prepared simply by diluting 10 volumes of glacial acetic acid with 90 volumes of distilled water. Store preferably in paraffined bottles with parafinned stopper.

(3) *Dilute Hydrogen Peroxide solution.* A freshly opened Merckozone should be used. 10 volumes of this diluted by 90 volumes of distilled water will roughly give 1% H_2O_2 solution.

It is preferable to prepare it freshly before use but it can be stored for 2 to 3 days in brown bottles in dark.

(4) *Standard blood.*—A sample of ·5 c.c. oxalated blood (too much oxalate should not be used) is used for determination of its iron content by Wong's method [6] and from that its hæmoglobin percentage is calculated. Any other accurate method for hæmoglobin determination, *e. g.*, oxygen capacity method may be employed. But our method of choice was Wong's as it required no special apparatus and their expert manipulation and requires relatively smaller quantities of blood. The standard blood is freshly obtained from an animal or it may be any surplus oxalated blood of a patient sent to the laboratory, for other determinations. We employed our laboratory sheep and its blood showed a remarkable constancy in its hæmoglobin content day after day. Bing and Baker[1] recommend use of standard blood solution which can be stored in a Frigidiare for a month.

Procedure

Collection of blood.—Blood is obtained by needle puncture from any finger tip—except the thumb and small finger—and should be freely flowing. Milking vitiates results and should be avoided. From the amount collected at the punctured spot, blood was taken into a white cell-counting pipette of a hæmocytometer graduated into 10 marks up to ·5 mark and blown into 10 c.c. of distilled water in a test tube. Water should be drawn into the pipette and blown back into the test tube and the process repeated several times. The blood is then thoroughly mixed with the water. This gives a dilution of the blood about 1 2000. Great care is required to measure such small quantities of blood and the success or failure of the method is primarily dependent on this. Lesser quantities of blood may be taken, if the amount of freshly flowing blood which has collected is not sufficient and blown into corresponding volumes of water (*e. g.*, ·4 into 8 c.c., ·3 into 6 c.c. and so on) but it must be remembered that smaller the quantity used, the greater is the chance of error unless special micropipettes are used. Therefore, as a rule, blood should be taken up to ·5 mark which is approximately only ·005 c. c. and with a puncture made by a thick needle, there will be seldom any difficulty in getting the requisite amount.

The standard blood is similarly measured and diluted in another test tube.

The Benzidine reaction.—½ c.c. each of the diluted standard blood and the sample is next pipetted by a Ostwald pipette into two graduated test tubes. Then is added 2 c.c. of Benzidine reagent into each from a microburette and the mixture thoroughly shaken. Next 1 c. c. of H_2O_2 solution is added from a microburette. These are thoroughly mixed and left for development of colour. At first a blue colour is obtained which slowly and gradually changes to red brown and the red brown colour goes on intensifying. We generally waited 30 minutes when about 80% intensity is obtained but if time is not pressing, one can wait up to 1½ hours for full development of the colour. Next the mixtures are diluted with 10% acetic acid solution up to 10, 15, 20 c.c. according to the colour developed and shaken. The mixture now looks more transparent and the colour changes to sepia.

The reading and calculation.—After 8 minutes of addition of 10% acetic acid and before 10 minutes —after 10 minutes the colours begin gradually to fade—the mixtures are placed in colorimeter cups and read. The standard is placed at 10-15-20 mark according to the intensity of the colour which, as previously stated, has a time factor and will depend on the interval allowed before addition of the 10% acetic acid.

The calculation is very simple. If A is the percentage of hæmoglobin in the standard blood

$$X = \frac{A \times \text{reading of Standard}}{\text{reading of sample}}.$$

Precautions to be taken and discussion of the Method.

1. The blood should be freely flowing and not squeezed. The measurement of the blood should be very carefully done and should not much overstep the ·5 mark when first drawn in.

2. The reagents and specially Benzidine reagent should be carefully prepared according to directions and stored. The Benzidine reagent should be colourless and no colour should develop if 2 c.c. of it is kept in contact with 1 c.c. of H_2O_2 solution for one hour.

3. The order of addition of reagents is very important and should be blood plus Benzidine plus shake plus H_2O_2 solution. The order mentioned by BING and BAKER, *i.e.*, Benzidine plus blood plus H_2O_2 solution, is not recommended because H_2O_2 solution may then come in contact with blood before it comes in contact with Benzidine and the intensity of the developed colour will be less.

4. *Time Factor.* The colour attains about 80% intensity in ½ hour and is maximum at 1½ hour and so the safest procedure will be to wait up to 1½ hour and then dilute with 10% acetic acid. But provided both standard and sample bloods are diluted with 10% acetic acid at almost the same time and read in the colorimeter between 8 to 10 minutes of dilution, there will be no error.

5. The test tubes to be used should be thoroughly clean as traces of blood would vitiate the results.

In preparing Benzidine reagent, the general procedure is to dilute the dissolved Benzidine with distilled water upto 100 c.c. but we accidentally discovered that if glacial acetic acid is used so that the reagent is a solution of Benzidine in glacial acetic acid and not in dilute acetic acid, the colour change is more intensive. So we will recommend use of such Benzidine reagent in case of samples of very low hæmoglobin content.

Again BING and BAKER recommend 20% acetic acid solution for dilution of the blood plus Benzidine plus H_2O_2 mixture but our experience is that 10% serves the purpose as well and is even better as the colour does not fade so rapidly as with 20% acetic acid.

We have already referred to the order of adding the reagents and in our opinion this is a better procedure than recommended by previous authors.

Accuracy of the method and its comparison with other methods.

First we tested the accuracy of the method itself in the following ways :—

1. Repeated comparisons of a sample of blood with a standard gave readings never varying by more than ±2%.

2. ½ c. c. of the same diluted blood was measured into two test tubes and reagents were added and the two compared in the colorimeter. The error was never more than ±3%.

3. Some sample of blood was compared to the standard animal blood in the usual way and its hæmoglobin was thus estimated. Next the iron content of the same sample of blood was estimated directly by WONG's method and hæmoglobin value calculated. The two figures were never found to differ by more than ± 5%. So we think we are justified in assuming that this method gives results correct within ±5%.

We next compared this method with the common methods. The same sample of blood compared by HALDANE-GOWER and SAHLI methods gave divergent results sometimes as high as 15%. When the same sample of blood was compared as regards its hæmoglobin content by this method and SAHLI, the difference was 5 to 10%, and that also sometimes in plus direction, othertimes in minus direction.

Therefore, again, we are justified in stating that this method is the more accurate of the three.

Hæmoglobin content in blood of young Indian adults as measured by this method.

In continuation of this work, we carried out hæmoglobin determinations by this method on 36 college students of ages between 15 to 23, average age being 17 years, and the results were as follows :—

1. Lowest 12·2. Highest 14·7 G. per cent.
2. Above 14 G. per cent—8.
   Between 13 G.% & 14 G. %—18.
   Below 13 G. % —10.

3. Average—13·55 G. hæmoglobin per 100 vol. The younger students showed relatively higher content. When we wanted to compare this figure with a standard we were at a loss. In the beginning of this century Haldane's figure was the only authoritative one, being 13·8 as calculated from his date of 18·5 c. c. O_2 per 100 volumes of blood. But recent workers have found higher values *e. g.*, Price Jones 14·5, Foster and Johnson 15·63, Haden 15·6 and Williamson 16·9 (7, 3, 4, 10). If we there fore take 15·6 to be the standard, blood of young Indian males according to our finding comes up to 87% of that standard. Again, if we take 4·5 millions to be the average R.B.C, count of Indians, the colour index comes to be $\frac{87}{4·5} \times \frac{5}{100} = ·96$.

Of course, we admit that we have made too few determinations to generalise and our group does

not include all ages and females and as such our figure is bound to be a little high.

A suggestion: In this connection we are tempted to quote the following lines from HAWK[6]

"It has been the custom among physicians to express hæmoglobin values in terms of percentages of some arbitrarily established normal .* * *. The result has been that at best half a dozen different concentration of hæmoglobin are accepted as normal 100% amongst practitioners. The logical way to avoid this chaotic state, it would seem, is to express hæmoglobin values in terms of actual concentrations without being committed to any arbitrary scale of normals."[6] It is therefore high time that diverse methods of hæmoglobin determinations in diverse arbitrary scale of normals should be done away with. The more correct methods should be adopted and by whatever method it is done it should express hæmoglobin values in terms of actual concentrations. The colour index may be calculated from the percentage amount of hæmoglobin. For example, if it is seen that the normal value for hæmoglobin is 15·6 and normal R. B. C. count is 5000000, that ratio will express the normal colour index 1. If then it is seen that Indian normal values are 13·5 and 4500000, Indian colour index will be $\frac{13·5}{4·5} \times \frac{5}{15·6}$ or ·96. We respectfully draw the attention of the profession to this suggestion.

Summary:

1. A simple micro method is described for estimating hæmoglobin in very small quantities of blood—which does not require any special apparatus.

2. The method is of value not only for quantitative estimation of hæmoglobin but may be employed for detection of minute quantities of blood and for forming a rough idea regarding the amount.

3. Hæmoglobin estimation of blood of a group of young Indians showed an average of 13·56 G. per cent, which quantity is in accord to the Indian figure of R.B.C. of 4·5 millions per c.m.m. of blood, if the colour index is near 1.

4. A suggestion is made to substitute the present chaotic state of hæmoglobin estimations in terms of arbitrary scales for the more scientific method of expressing hæmoglobin values in terms of actual concentrations. Calculation of colour index will not be difficult if this new suggestion is adopted.

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Longevity and Arteriosclerosis*

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MR. PRESIDENT, LADIES AND GENTLEMEN,

My subject to-day is Longevity and Arteriosclerosis. Prevention of epidemics, and advance of sanitation have prevented death in large numbers; advancement in the knowledge of infant feeding has lessened infant mortality in civilised countries, but we have not far advanced in bestowing the gift of a healthy long life on individuals and nations. People are afraid of increase in population, and that an advanced old age will lead to famine in future due to scarcity of food; but as science progresses, scientists will provide us with food in tabloid form, as it is already being investigated and prepared from cotton, to give us enough calories and vitamins too. Selection of right and healthy parties to marriage will give us healthy offsprings, and birth-control and sterilisation of unfit under state regulations, will see to creation of healthy and good stock in future. We find the age of 50 and after, is the age from which point rapid deterioration of health occurs, and deaths occur due to cardiac failure, apoplexy or renal failure. Very few survive to the age of seventy, eighty, ninety or a hundred years.

If we examine clinically these octogenarians and others, we find that comparatively their arteries are softer and less degenerated, the tension is correct, the pulse is quieter and the systolic and diastolic pressures are not high. On enquiry from my adult patients of 50-60 with normal arteries and systolic tension 125 to 130 m. m. Hg. or lower, I find that there is a history of family longevity in both the parents and grand-parents, provided none of them have been carried away at an early age by accidents of life, as motor car or railway accidents or by infectious diseases, such as pneumonia, enteric fever etc. This condition of healthy arteries from birth is essential to a long life.

The healthy condition of arteries in persons with long lives is inherited, and consequently the comparatively low pressure in them is also inherited. I have found that excesses in food and drink which would in ordinary individuals raise the blood pressure markedly, raise it by a few millimeters. in these fortunate individuals. In the former the patients suffer from symptoms of high blood pressure, in the latter the patient, feels a little unwell but soon feels better after a purge or slight restriction in diet.

The reason why some people in spite of excessive drinking and eating have lived to an advanced age, is because they have inherited good arteries and an equable blood pressure through the balanced action of healthy endocrine organs.

Some centenarians give history of moderation in eating and drinking, others do not; the explanation for the latter is, good heredity. From this we learn why people fail to attain a healthy old age; it is because we have always begun to treat the cases at the wrong end of life. The time has passed and the body is beyond redemption. At that time we are tinkering, putting a patch here and a patch there, now treating the kidneys, at another time the heart and at another time the cerebral symptoms.

Besides are we sure that the drugs we use, bring about the desired effects of antagonising the ill effects of arterial degeneration in different organs?

We use drugs of vasodilator groups to lower the blood pressure and treat the symptoms as angina pectoris, cerebral encephalopathy and uraemia. Are we satisfied with their results? We are not; otherwise we would not be using numberless drugs from the pharmacopœia and those put before us by various firms of chemists. I will here quote briefly from *The Lancet*, the observation made by EVANS and HOYLE on 90 ambulatory hospital patients suffering from angina pactoris, excluding syphilitic etiology. The essential criterion was a recurrent pain on effort behind the sternum usually, radiating across the chest and sometimes in other directions. The difficulty of adequate control was provided by prescribing a placebo for a period of 2 to 4 weeks, before any drug was tested and repeating the control period later as often as was considered necessary. The patient kept notes of number and severity of attacks; the following drugs were tested: sodium, nitrite, mannitol hexanitrate, chloral, morphine, papaverine, phenacetin, diuretin, calcium diuretin, euphyllin, belladonna, digitalin, lacarnol and harmol. In order to avoid suggestion the drug was prescribed in mixture form

* Paper read at the tenth All India Medical Conference, 1933.

when possible. An analysis of the results shows that placebo gave better results than most of the active drugs and appeared statistically the better form of treatment. The results of this investigation which though negative are none the less valuable, and will not surprise many who see much of angina and will confirm the belief that regulation of mode of life is still the most important part of treatment. These drugs not only fail to improve the condition of angina pectoris but fail to lower the blood pressure and improve the symptoms due to arteriosclerotic condition of cerebral and renal vessels. In spite of all kinds of remedies taken internally the patient proceeds to meet his doom; finally the crisis occurs, the cardiac circulation begins to fail, or apoplexy occurs or renal failure sets in. Now injections come in to patch the hole, which has become wider. Digitalis, camphor cardiazol for heart failure, venesection, purging, lumbar puncture for apoplexy, and glucose injections etc. for renal failure are resorted to. As I am on this point of treatment of arteriosclerosis and as it is our duty to prolong life by all means in our power, I wish to discuss a point on which high authorities differ. Some physicians are against lowering the blood pressure which fortunately does not get much lowered by the drugs which we are using. They say, the body maintains the high pressure because it needs it for proper circulation; and though the systolic, be 175 m.m. or 200 m.m. as the patient feels well it need not be lowered. Others are for lowering it by all means in their power. To me the golden mean is the best form of treatment. A patient may not feel for months the bad effects of high blood pressure, but the latter damages the heart and blood vessels, and he may die suddenly of apoplexy or cardiac failure, because under the strain of high pressure the cerebral vessels or the heart muscle may give way. The pressure to maintain is not age plus 100 but half the age plus 100 *i. e.*, a man of 60 should have a blood pressure of 130 or 135 to live a moderately comfortable life. Too much lowering of blood pressure would lead to cardiac failure or renal failure, and for all this the sphygmomanometer is our best guide. As I said previously we begin our treatment at the wrong end of life. Senile changes are bound to occur in the arteries but they are slow and take a long time, so that if proper precautions are taken in the beginning most of us ought to die a peaceful and quiet death by sudden stopping of the central pump—the heart, from its exhausted nutrition, and death would be like sleeping in this world and waking in the next.

But unfortunately the teachings of our ancestors have taken a firm root in us. The patient comes, he is examined and the immediate next step is writing a prescription; in this way thousands of tons of medicine must have filled the stomachs of patients the world over and with what benefit? Comparatively very little. Tons of medicines are prescribed giving no benefit except as a placebo; but plain water would be a far better placebo, not damaging the sensitive gastric mucous membrane. Liq. Ammon. acetatis is prescribed by tons in fever mixtures; how many have noted it to cause diaphoresis. As if these powders and tinctures were not sufficient, for the last few years have appeared on the stage, patent medicines manufactured by pharmaceutical chemists of known and unknown reputation. It reflects on our credulity to use inert and expensive drugs in spite of our considering ourselves level-headed men. We have not the moral courage to refuse base imitations. We prescribe any and every drug the agent asks us.

For our reputation and the health and purse of the patient it is incumbent on us definitely to refuse to prescribe any preparation even from reputable firms which is not backed by statistics of a hundred or thousand patients treated in a well known Hospital under the direction of a well known physician. By this method only we shall have well tried and efficient remedies; and scientists and well known chemical firms will have the stimulus to bring out effective remedies when they find that unfair competition from unknown and unreliable firms throwing valueless drugs on the market have been eliminated. As I am on this subject I might give out what is in my mind about our failures in treatment. Take a case of pneumonia and read an article on its treatment in any well known book on medicine or treatment. We will find a host of drugs mentioned one after the other, some as acting against the cause, others to treat symptoms. What does that show? It shows our incompetence to treat it; whenever there are numbers of drugs recommended to cure a disease, believe it we are on uncertain grounds and the chances are fifty-fifty, nature curing it or the disease over-powering, we acting as active meddlers. A physician has noted that he suffered from pneumonia thrice, he suffered more and longer when he took treatment for the disease on two occasions, on the third occasion without treatment he became well soon. Now coming back to the treatment of pneumonia we have sera, vaccines, immunogens, phylacogens, sodium nucleinate, salvochin etc. to cure the disease, and digitalis, camphor, cardiazol and glucose injections for

sustaining the heart; are we satisfied with them? No, then why should not our big hospitals in England, on the Continent, in America and India, try in 100 cases each of these drugs and give us the results of treatment so that the valueless ones may be eliminated and those which show the best results may be tried by the new and old physicians alike. We do not gauge the hopelessness in which our practice has fallen. Sera, vaccines, immunogens, sodium nucleinate and salvochin all have been tried without any benefit. Meanwhile the patient tides over the crisis and lives or dies. We have done much but we have neglected much through ignorance, self-complacency or indolence and blind faith in past traditions. The next generation will look with amusement and wonder at our belief in powders and tinctures and extracts most of which are inert. The students and physicians of the coming generation will look with amusement at the antiquity of our books on medicine, materia medica and treatment. There will be a museum installed to show all the medicines that are inert and useless, which in our fond belief of their efficiency we used, to cure disease but failed. The uselessness of our drugs I can well point out by taking from the Materia Medica the example of iron and its preparations. There are 15 and more listed, out of which latterly we have found out Ferri et Ammoni Citras to be the best. Can't we do the same with all other preparations? Friends, I have pointed out in brief the ways we should proceed. We have advanced satisfactorily in pathology, bacteriology, biochemistry but our research in treatment of diseases falls far short of the advancement in other branches.

Patients do not want to know the pathology and bacteriology of pneumonia. They want to recover from it. What avails to know the type of pneumococcus if the patient does not survive? What avails the knowledge of increase of urea and uric acid in blood, if the patient does not recover from uræmia? I think a batch of scientists in every country should devote all their time in research of finding new remedies, put forth after a thorough trial on hospital patients. We have not had ten ways of operating for appendicitis; why should we then have ten drugs put forth for treating pneumonia or other diseases. Now coming to my subject of arteriosclerosis our aim and object should be to tackle it from young age. The degeneration of the arteries is slowly proceeding from the young age, due to wear and tear which every tissue of the body suffers from; our aim and object should be to prevent excessive and rapid degeneration of the arteries and secondarily of the various organs and hormone-producing glands supplied by them; how could this be done? This could be done by proper nourishment supplied to the body through the circulation of healthy blood.

For this healthy blood we have not to think of drugs but of the food supply and regulation of mode of life. The diet should be a balanced one with minimum of proteins and fair amount of vitamins. The mixed diet which we ordinarily eat is sufficient to supply all our wants but it should not be in excess. Excessive amount of food kills more than excess of drink, because the excessive amount of food being not properly digested throws its burden on the stomach and secondarily on the liver and then on the kidneys. The liver metabolism suffers and, therefore, it is unable to neutralise all the toxins, the immature products of protein metabolism enter the blood, the toxæmia increases, the toxin acting on the intima of the arteries and also causing degeneration of the media through the vasa vasorum. This degeneration of arteries leads to lessening of blood supply to the various organs and internal secretory glands resulting in an atrophy of the essential cellular elements and replacement by non-essential fibrous tissue. Thus is precipitated premature decay and old age. The diseases following in on the wake of arteriosclerosis are many, a few of which can be enumerated. Beginning with the heart and blood vessels, it causes angina pectoris, coronary thrombosis, cardio-sclerosis, arteritis, aneurysm. In the brain it leads to cerebral hæmorrhage and thrombosis, and due to atrophy of cells, loss of memory.

In the kidneys it causes degeneration and atrophy of glomeruli and tubules and secondary uræmia. In stomach and intestines, it leads to atrophy of mucous membrane and secondarily causes dyspepsia and diarrhoea. In pancreas, it leads to diabetes. In testes and ovaries, it causes atrophy of essential cells and secondary senile changes in the body. Our duty is to prevent all these by a careful watch from the young age, and not to tinker when the parts of human machinery are broken here and there beyond repair. Here I am reminded of certain lines by Poet Sadi which mean "the source of a rivulet you may perhaps hold with a needle, but when it is full you may not be able to hold it with an elephant." It is an apt simile for our subject of arteriosclerosis and longevity; by a simple regulation of mode of life and proper dieting you may avoid degeneration of arteries and live a long life, whereas by carelessness you only hasten your death. Excessive drinking of liquids, both alcoholic and non-alcoholic, increases the volume of blood and puts extra strain

on the heart and blood vessels. For this very reason the indiscriminate advice to drink large amount of water to flush the kidneys, especially to old people is fraught with danger. The intake of liquid must be of the same amount as the output of urine, otherwise, the heart, the vessels of the brain and kidneys are embarrassed. We see many old Pathans and old Chinamen, it is because due to scarcity of food in their countries they are spare eaters. The medicine of the future will be a preventive one; patients will come once a week or so to be examined and advised about their health; they will be advised not to take this or that drug but they will be asked to cut out, this or that item from their diet which does not agree. Drugs for treatment of arteriosclerosis are failures; but I have found one drug containing colloidial silica with extract of drosera going under the trade name of Drosil to be of some benefit in relieving the symptoms of arteriosclerosis when to be powdered and taken internally. The treatment and prevention of arteriosclerosis are the same; cut down the amount of diet, especially the protein, next the starch and allow moderate amount of fluid. No excessive tea, coffee or cocoa or nuts should be allowed. Relaxation from mental strain is highly essential. Regarding the way to a long and healthy life, our suggestions should be the proper intake of good food in moderate amounts, moderate intake of liquids, and avoidance of infectious diseases, even common cold upsetting the balance of health and for the time being making the circulating blood unhealthy. With proper attention to diet from young age, proper attention must be paid to moderate exercise, excess of which strains the heart. To those who cannot take exercise walking in the open air is as good. Proper food, pure water, pure air and moderate excercise lead to a healthy old age, as our forefathers used to live from experience which we fail to follow in spite of our knowledge. The boasted polyhormonal remedies, and the tonics advertised by Hakims and Vaids should be prohibited from being sold, by legislation. In old age they are positively harmful as they increase the blood pressure and precipitate angina pectoris, apoplexy or uræmia; they precipitate the very diseases they are out to cure.

Before the government can take steps to prevent these noxious remedies from being sold, it is our duty to prevent patients from poisoning themselves by being hypnotised into buying and taking them after reading fraudulent advertisements.

Summary

(1) Prevention of old age is by prevention of arteriosclerosis.

(2) There are no drugs for the treatment of arteriosclerosis, once it has taken a foothold.

(3) For longevity and good health, a good heredity and moderation in food and drink, pure air, moderate exercise, and avoidance of worry are essential.

(4) The right time for preventing arteriosclerosis is young age; once it has supervened, moderation in food and drink may minimise its banal effects but it goes ahead all the same to destroy life.

(5) An intensive research into treatment, is highly necessary and it is the bounden duty of physicians all the world over to concentrate their attention on bringing out well-tried and successful remedies; at present we have a plethora of half-tried remedies.

(6) It is the duty of practitioners to prescribe remedies put forth by well known chemical firms, which have been recommended by hospital physicians of known reputation, after thorough trial.

(7) It is the duty of practitioners to instruct their patients how to live a long and healthy life, through well-balanced diet, and to prevent them from poisoning themselves, by taking quack remedies and so-called rejuvenating nostrums; it is also their duty to anticipate diseases, and prevent them.

JOURNAL
OF
The Indian Medical Association

Vol. III APRIL No. 8

Editorial Comments

Chronic Amoebiasis

It is a common knowledge amongst the medical practitioners of a tropical country like India that amœbic infection is an important cause of a considerable degree of morbidity amongst the people. Of course the usual manifestations of the infection are acute dysentery with passage of mucus, blood and pus in the stools. But this acute phase sooner or later passes into the chronic stage in absence of efficient treatment. CHOPRA reports that a routine examination of the stools of patients in the Carmichæl Hospital for Tropical Diseases, Calcutta, has shown an incidence of 12% of amœbic infection. Cases are not infrequent where the infection has at no time shown any acute symptoms. It is chronic or latent from the beginning. Such cases of chronic or latent amœbiasis constitute the carriers who are the chief sources of spread of the disease, by means of contamination of food and drink with the encysted forms of *E. histolytica*.

The only way to the control of amœbic infestation in a community is to spot out the acute, the chronic, and the latent cases as early as possible and to institute the most efficient anti-amœbic treatment till a complete cure results. It is also essential that contamination of food and drink through contact with the excreta of carriers must be prevented. The latter is a public health problem involving an adequate treatment of the sewage before its final disposal. The attention of the medical practitioners should, however, be focussed on the problem of early recognition of *amœbiasis* and its proper and efficient treatment. The early diagnosis of *chronic amœbiasis* will depend upon the physician's suspicion of its presence in cases of vague abdominal discomfort with or without definite pain, flatulent dyspepsia, diarrhœa, hepatic enlargement with tenderness, and on the thoroughness of his search for the vegetative or the cystic form of the parasite in stools. A word of warning would not be out of place here to point out that in some cases the presence of *E. histolytica* cysts in stools may be a mere coincidence and not the cause of the aforesaid symptoms. It should be emphasised that the manifestations of amœbiasis are protean. Duodenal ulcer, cholecystitis, and pulmonary tuberculosis may be simulated. Abscesses of the liver and the lung are not infrequent complications, though they are very rarely seen in the brain, the spleen, and the kidneys.

The diagnosis of amœbiasis should be confirmed by the presence of *E. histolytica* in stools, a specimen of which should preferably be obtained after the administration of a saline purgative such as magnesium sulphate and be examined as soon as possible after evacuation at body temperature. In some cases clinical diagnosis may be made from a visualisation, through the procto-sigmoidoscope, of typical white mucus-capped small round ulcers on the mucous folds of the rectum and the lower sigmoid with intervening areas of healthy mucous surface and it may be confirmed by an examination of a smear from these ulcers for the presence of *E. histolytica* cysts.

In other cases an examination of the cæcum and colon by opaque barium enemata may reveal characteristic patchy deformities in the region of the cæcum or irritability and hyper-peristalsis of the terminal ileum and cæcum. In a suspected case of chronic amœbiasis these modes of investigations are essential, otherwise many cases of early carcinoma of the bowels and chronic non-specific ulcerative colitis may escape detection. If in spite of negative bacteriological and radiological findings, a diagnosis of chronic amœbiasis be justified on clinical grounds, the appropriate treatment may be continued for at most a fortnight. Absence of definite clinical improvement warrants a revision of the diagnosis.

The treatment of amœbiasis is a complex problem which has been baffling all attempts at solution till to-day. The numbers of drugs advocatted from time to time have been near about a dozen. Time has come when the medical profession should take stock of their relative curative properties. There is no doubt that some great advances have in recent years been made in the therapeusis of *amœbiasis* but much still remains to be done. There is almost universal agreement that *emetine hydrochloride* strongly advocated by ROGERS in 1912, is the drug of choice in acute amœbiasis. But the accumulated experience has

shown that only a third to a half of the patients are cured by emetine, whereas the remainder passes into the chronic stage. The reasons are that (i) *emetine* is a toxic drug which cannot be used in sufficient dosage for a long time for fear of myocardial injury and peripheral neuritis, (ii) it fails to exert its action on the *amœbae* lying deep at the base of the ulcers, and (iii) the acid reaction of the intestine interferes with the efficient action of emetine. Hence other drugs had to be tried. *Emetine bismuth iodide* was therefore introduced for oral administration. It is also a toxic compound which fails to eradicate the infection in a large number of cases in spite of two or more courses, as the reports of KNOWLES from Calcutta, and of WILLNER from China indicate. Next the organic arsenical preparations such as *stovarsol*, *treparsol*, and *arbarsone* were recommended for oral use. Clinical trials have definitely demonstrated a higher percentage of cure with arsenic preparations in combination with emetine than with the latter alone. But the danger of arsenic poisoning in 3 to 4 per cent of cases as pointed by BROWN should be remembered. Toxic erythema is the commonest complication though exfoliative dermatitis and peripheral neuritis do occur under stovarsol. Treparsol is too rapidly eliminated to give rise to peripheral neuritis. Carbarsone has been reported to yield so far very promising results without any untoward effects. CHOPRA has recently studied the action of this compound in a series of 31 cases of chronic intestinal amœbiasis in doses of $3\frac{3}{4}$ gr. in gelatin capsules twice daily for a period of ten days, and has reported 74·2 per cent cures. He also points out that the proportion of probable cures to failures in this series was 5·75 : 1 as compared with 3·5 : 1 obtained by KNOWLES in a similar series with *emetine* bismuth iodide and 3·16 : 1 obtained by ACTON and CHOPRA and with *kurchi* bismuth iodide. In view of such striking results further trials with *carbarsone* are urgently required.

Again, some quinoline derivatives, *e. g.*, *yatren*, which contains 28% iodine and *vioform* which contains both chlorine and iodine, have lately come into prominence. Yatren in doses of four 0·25 G. pills thrice daily for 15 days has been claimed by BIGGAM to have given an apparent cure rate of 72 p. c. The only untoward effect of yatren in such large doses is diarrhoea. Moreover, it is a very costly drug. LEAKE and others claim that vioform administered in doses of 0·75 G. daily for 10 days and repeated a second time after an interval of a week is better than yatren. Besides, vioform does not give rise to intestinal irritation and diarrhoea. Still more recently another drug—a derivative of ipecacuanha—gavano by Messrs. Bayers & Co., is on the market. CHOPRA concludes from the results of his clinical trials with this drug that it may be used orally without any toxic effect but its amœbicidal property is much less than that of emetine bismuth iodide, kurchi alkaloids, and carbarsone, the cure rate being only 33·4 p. c. He, however, points out that in cases of amœbiasis with hepatic complications where carbarsone and kurchi alkaloids are completely ineffective, gavano may be tried.

Lastly, we draw the attention of the medical profession in India to the outstanding fact emerging out of the analytical study of the reports of the various workers mentioned above that carbarsone is probably the least toxic but the most effective drug for chronic intestinal amœbiasis without hepatic involvement. We hope, that this compound would be given further clinical trials on an extensive scale under controlled supervision, with a view to the assessment of its proper place amongst numerous anti-amœbicidal drugs.

Current Medical Literature

MEDICINE

The Prognosis of Heart Disease

R. T. GRANT (*Bulletin of the New York Academy of Medicine*, Second Series, Vol. IX, No. 8, August 1933), summarises his conclusion as under :—

1. The after-histories of a group of 1,000 men suffering from heart disease have been followed for 10 years.

2. Only 13 of the 1,000 remain untraced; the after-histories of 70 per cent, throughout the period, are completely known.

3. The diagnostic criteria in the classification of the cases are defined. The cases are classified chiefly from two aspects: (1) valve lesion, (2) exercise tolerance and cardiac enlargement.

4. Autopsy reports are available for 142, or one-third of the deaths. Comparison of clinical diagnosis with post-mortem findings shows that diagnosis of valve lesion is in general accurate. Between the clinical estimate of heart size and the weight of the heart after death the agreement is sufficiently close.

5. The after-histories are characterised mainly by the following features :—

(*a*) 51 per cent are known to be alive. Of these survivors at least 42 per cent have lived uneventually throughout and with changed physical signs.

(*b*) Auricular fibrillation, present originally in 10 per cent, develops later in 8 per cent.

(*c*) Subacute bacterial endocarditis, present initially in 7 per cent, develops later in 5 per cent.

(*d*) 47 per cent are known to have died.

6. Correlation of after-history with the factors used in the classification shows that after-history is to be foretold not so much by the presence or absence of valve defect or by the type of defect but more closely by the degree of cardiac enlargement and by the grade of cardiac failure as estimated first by the exercise tolerance and secondly by the presence of venous congestion.

7. Prognosis is most satisfactory, when based on cardiac enlargement and cardiac failure and is modified by associated findings.

8. On this basis the chief prognostic indications are :—

(*a*) *Good prognosis.*—Cases with little or no enlargement and good or fair exercise tolerance. Only about one-fifth of these cases die; almost a half of them live uneventually and unchanged.

(*b*) *Poor prognosis.*—Cases with moderate enlargement and poor exercise tolerance. Half die within 10 years.

(*c*) *Bad prognosis.*—Cases with great enlargement or congestive failure. Few survive. The average life in the presence of venous congestion is 2½ years.

9. The chief factors modifying these prognostic indications are :—

(*a*) sub-acute bacterial endocarditis. The average life in its presence is 6 months. It develops mainly in cases of non-syphilitic aortic regurgitation (10 per cent.)

(*b*) the onset of auricular fibrillation influences unfavourably the general prognosis; the outlook in cases of established fibrillation depends on cardiac enlargement and exercise tolerance. In cases of congestive failure its presence influences favourably the immediate prognosis; the average life is increased to 5 years. It develops mainly in cases of mitral stenosis (20 per cent.)

10. Prognosis within the groups of cases with aortic regurgitation, mitral stenosis, etc., is discussed for each group separately. Prognosis is least favourable in aortic stenosis and syphilitic aortic regurgitation. There is no material difference in the death rate in those with non-syphilitic aortic regurgitation, with mitral stenosis, or with those lesions combined.

11. The general outlook for cases of valve defect is not so bad as is generally thought, even for syphilitic aortic regurgitation. The average life in cases with aneurysm is over 5 years and in its absence only 58 per cent of those with syphilitic aortic disease die within 10 years. Treatment with arsenic and mercury slightly but definitely prolongs life, treatment with potsssium iodide does not. *J. C. B.*

Chronic Tobacco Poisoning

F. CRAMER (*Münch. Med. Wschr. Ref. Med. World. Feb. 16, 1934*) takes a grave view of the dangers of using tobacco in any form. Beginning with the statement that *nicotine* is as poisonous as cyanide of potassium, he asserts that a hundred cigarettes smoked in the course of a day are almost certainly fatal and points out that the blood of a regular smoker causes a leech that sucks it to die in convulsions. Angiosclerotic dyspepsia is the commonest result of smoking (especially of ciagrs in which most of the nicotine remains in and can be recovered from the stump), leucoplakia, cardiospasm, achylia, and hiccough being frequent. Spasms of the sphincter and eventually leading to hæmorrhoids, often arise in tobacco addicts, while intestinal ischæmia causing spasms, meteorism and alternate conditions of constipation and diarrhœa with offensive motions are not uncommon. However, CRAMER admits that the personal factor may modify these troubles considerably and states that he himself is unable to smoke more than two cigars. *J. C. B.*

PEDIATRICS

Parasites of Kala-Azar and their distribution in the body

M. N. DE in his studies on the Parasites of Kala-azar and their distribution in the body (*Ind. Jour. Med. Res.* 21 : 627. 1934) has described the different technical methods for the demonstration of the parasites in kala-azar and other leishmania infections and has discussed their relative values. He showed that the parasites are always found in large number in the liver, spleen and bone-marrow and only occasionally in the other parts of the body. The parasites do not remain as freely circulating bodies but are always inside large swollen phagocytic mononuclear cells, the reticulo-endothelial cells of Aschoff or the clasmatocytes of Ranvier. Presence of parasites inside vascular endothelium could not be verified. When parasites are present in areas other than the liver, spleen and bone-marrow there is usually an inflammatory reaction excited by some cause. The relationship of inflammatory reaction to the infection of the reticulo-endothelial cells by leishmania has been discussed. *K. C. C.*

Investigation on Glycogen Disease

S. VAN. CREVELD described the results of new investigations performed in a boy with hepatomegaly and a particular disturbance in carbohydrate metabolism, formerly regarded as being the expression of an accumulation, especially in the liver, of glycogen which could only be mobilized with difficulty. The investigations in a second case (a girl) with hepatomegaly and the same deviation in metabolism are also reported. The clinical history of this girl is given in detail.

The glycogen metabolism was specially studied in both patients. A method is given of determining the glycogen in 1 c.c. of blood. The question of the cause of the difficulty in splitting glycogen in glycogen disease in general was studied and is discussed. Some new facts of *hepatomegalia glycogenica* were established and their value for the differential diagnosis from liver cirrhoses in childhood is stressed.

The distribution of glycogen in different organs and the stability of this glycogen in the heart was investigated in a case of *cardiomegalia glycogenica* and in a case of hypertrophy of the heart due to a patent septum ventriculorum. *K. C. C.*

Tuberculous Mastoiditis in children

H. R. SOUPER (*Proc. Roy. Soc. Med.*, 26 : 1585. 1933) treated 36 cases and his conclusions are :—

1. Tuberculous otitis media with mastoiditis is not uncommon in children aged under 2 years.
2. It is frequently unrecognised both by parents and practitioners and the dangers are not appreciated. The mortality is at least 50%. Further education is essential.
3. The earlier the disease is submitted to treatment, the better are the patient's chances.
4. The younger the child, the more fatal is the disease.
5. It is impossible to lay down hard and fast rules as to the most suitable time for operation and the extent of operation. Each case must be considered on its merits and the determining factors in prognosis would seem to be the age and the general condition of the child.
6. The simpler type of operation, where that appears to be sufficient, gives the best chance of recovery. Extensive operation, if found necessary and advisable, should be performed in stages. The cortical operation gives a recovery rate twice that of the radical. *K. C. C.*

Treatment of Alimentary Toxicosis

O. Saxl (*Acta. Ped.*, 16 : 542. 1933) following the suggestion of Schiff, who found considerable diminution of glycogen reserve in the liver of experimental animals in alimentary toxicosis, used a mixture of whey and 10% rice-gruel with 6 to 8% sugar. This diet was used in 12 cases of which 9 were cured and 3 died. This has two advantages (1) rapid improvement of general condition, and (2) retention of the water in the tissues. The only disadvantage is that in replacing it with the usual maintenance diet serious complication may arise. *K. C. C.*

Association Notes

INDIAN MEDICAL ASSOCIATION (Bengal Branch).

At the third Annual General Meeting of the Indian Medical Association, Bengal Branch, held on Wednesday, the 28th March, 1934, at 7 P. M., at the Association Hall, the following office-bearers have been elected for the year 1934 :—

President—		Sir Nilratan Sircar.
Vice-Presidents—	1.	Dr. C. C. Bose.
	2.	Dr. K. S. Ray.
	3.	Dr. J. N. Bose.
	4.	Lt. Col. K. K. Chatterji.
	5.	Dr. N. R. Sen Gupta.
Secretary—		Dr. Girin Mukherji.
Asst. Secretaries—	1.	Dr. Tarak Nath Ghosh.
	2.	Dr. P. C. Chakraborty.
Treasurer—		Dr. A. K. Chakraborty.

Medical Associations, Societies, etc.

Medical Societies, Association, Unions etc. are invited to make use of this Section of the Journal. —*Editor*

ALL-INDIA OPHTHALMOLOGICAL SOCIETY

The third Conference of the All-India Opthalmological Society was held in the Auditorium of the All-India Institute of Hygiene and Public Health, Calcutta, from the 19th to the 21st Dec., 1933. The attendance was the largest on record and was well supported by large numbers of delegates from all parts of India. Dr. A. FUCHS from Vienna and a large number of other visitors attended the Conference.

The Conference was formally opened by Sir HASSAN SUHRAWARDY, M.D., F.R.C.S., Vice-Chancellor of Calcutta University, in an excellent speech in which he pointed out the absence of facilities for ophthalmological work in India and regretted that this vast country was still very backward in the number of well-equipped and modern ophthalmic hospitals. The enormous ophthalmic centres in the capitals of the various provinces of India were maintained at Government expense, and in each magnificent work has been and is being done, but he hoped that wealthy Indian gentlemen would come forward with sufficient money for the establishment of more ophthalmic hospitals and schools where sufficient post-graduate training and research could be carried out.

In welcoming the delegates, Dr. D. N. MAITRA, Chairman of the Reception Committee, said that the problems of health which included that of the eye were at the bottom of social education and economic problems.

Lt.-Col. J. N. DUGGAN (Bombay) was formally proposed to the Chair by Lt. Col. E. O'G. KIRWAN (Calcutta) and seconded by Dr. G. Zachariah (Madras).

Lt.-Col. DUGGAN after acknowledging the honour paid to him in being elected to the office of President, gave in his address a comprehensive survey of the progress which ophthalmogical science has made in recent years and stressed the need for a well organised and sympathetic campaign for the prevention of blindness in India, as much of the blindness is preventible and yet there are enormous numbers of blind and partially blind people all over the country. He pointed out that India has earned a place in ophthalmology in the past. The intracapsular method of cataract extraction and sclero-corneal trephining for the relief of glaucoma have been two notable contributions to operative surgery, and acknowledged the work done in the clinical, pathological and operative lines by distinguished members of the Indian Medical Service who have gone far to raise the status of Indian Ophthalmology and pleaded this work must be continued hereafter if the prestige of Indian Ophthalmology is to go still higher. In concluding, he reiterated that as there was such an abundance of clinical material available in all parts of India, research should be encouraged on thoroughly scientific lines.

In the afternoon the scientific session was opened by a paper on epidemic superficial punctate keratitis in Bengal by Lt.-Col. E. O'G. KIRWAN. After describing in detail the clinical findings, pathology and treatment, he pointed out that the name superficial punctate keratitis was a misnomer, as in a large number of these cases the affection produced a mild iridocyclitis. He, therefore, favoured the suggestion of Lt.-Col. R. E. WRIGHT that it should be called *keratitis diversiformis et uveitis anterior*. The paper was illustrated by slit lamp paintings. An interesting discussion followed in which Drs. FUCHS, BHADURI, MUKHERJI, SRINIVASAM and Col. DUGGAN took part.

An interesting paper on the treatment of spring catarrh by injections of foreign proteose emulsion from the urine by Dr. BANAJI (Bombay), produced a lively discussion in which a large number of members took part.

Dr. FUCHS (Vienna) read a paper on the surgical treatment of iritis.

This was followed by a paper on Parinaud's conjuctivitis by Dr. SANYAL (Calcutta) and was illustrated by lantern slides.

The afternoon session closed with a paper by Dr. BHADURI (Calcutta) on observation on the leprotic affections of the eye in which he pointed out the causes of the wide variation of the ocular incidence in leprosy and advocated the benefit of iridectomy in the early stages of iritis.

In the evening a magic lantern slide demonstration followed by a cinema were given by Lt.-Col. E. O'G KIRWAN, on prevention of blindness and care of the Eyes in India.

The second scientific session was held on the morning of the 20th December 1933, and among the most interesting papers was one by Dr. S. K. MUKERJI (Calcutta) on further observations in glaucoma as a result of epidemic dropsy which

comprised tonometric, ophthalmoscopic and visual field studies. An interesting discussion followed in which Lt. Col. E. O'G. KIRWAN, Drs. BHADURI, NARAYAN RAO, GNANADIKAM Dr. N. CHATERJI took part.

The President of the conference Lt.-Col. DUGGAN read a very instructive paper on the treatment of external eye diseases with ultra-violet light. A discussion followed in which many of the members took part.

Dr. J. GNANADIKAM'S paper on iridenclesis was most instructive and he pointed out the advantages of this operation over other methods of decompression from his experience of a large number of cases. In the discussion which followed, Col. DUGGAN and Drs. BANERJI, SRINIVASAM and R. GHOSH took part. It was clearly evident that the operation of iridenclesis has many advocates in India, although most ophthalmologists favour sclerocorneal trephining in chronic primary glaucoma in the majority of cases.

An interesting paper which stirred up a hornet's nest was one by Dr. C. N. SHROFF on the intracapsular cataract operation suitable to the conditions in India in which he stated from his personal experience that the expression operation of Lt.-Col. SMITH with certain modifications is the most suitable to the conditions in India where there is such a large amount of work to be done in a limited time. He pointed out that in all cases the lens should be tumbled but cases in which there was a strong zonule, capsulotomy should be done. A spirited discussion followed in which Lt. Cols. DUGGAN, KIRWAN and Drs. FUCHS, NARAYAN RAO SRINIVASAM and others took part.

The Madras School of Ophthalmology produced a most interesting paper by Lt. Col. R. E. WRIGHT, and his staff on von Hippel Lindau's disease. There were only three cases seen out of more than a quarter of a million fundus cases examined in Madras. Two of the cases were followed up for a long period; in all cases the changes observed were only in the retinal vessels on one side and there was no evidence of a familial nature, nor of the central nervous system or any skin affections.

The third scientific session took place on the afternoon of the 20th December 1933 and among the most interesting papers read was one by Dr. BHADURI (Calcutta) on some observations on hypotension in cholera in which he showed tonometric records in 46 cases and that the blood specific gravity varies inversely with the intraocular tension. Capt. K. SEN (Calcutta) read a paper on asepsis on the conjunctival sac in intraocular operations, in which he stated that 64 per cent of the conjunctival sacs before intraocular operations showed no growth in 48 hours' culture, and of the remaining 36 per cent, one-half showed harmless organisms and the remainder streptococci, staphylococci, etc. He advises the use of mercuro-chrome drops one per cent, irrigation of the conjunctival sac with one in 10,000 oxycyanide of mercury and immediately before operation, irrigation of the sac with normal saline. A lively discussion followed in which many members took part.

Perhaps the most interesting paper in this session was read by Capt. S. C. DUTT (Calcutta) on the development of modern ophthalmology in Bengal in which surveyed the period extending from the time of the Honourable East India Company in 1796 to the present day.

The fourth scientific session took place on the morning of the 21st December and among the most interesting papers was one by Lt.-Col. E. O'G. KIRWAN on blue sclerotics in which he pointed out the paucity of cases in this vast country. He illustrated two cases with coloured sketches and discussed the different views on the causation.

Dr. RAFATULLA'S paper on Webster's operation for entropion produced a keen discussion and the author advocated this mucous graft operation as a fool proof method in the cure of this comomon and troublesome complaint.

Dr. H. K. INDRA (Calcutta) read three short papers on lenticonus posterior, Kruckenburg's spindle and a case of aphakia with apparent accommodation. All these papers were beautifully illustrated with slit lamp paintings carried out at the Eye Infirmary, Medical College, Calcutta.

The fifth scientific session took place in the after-noon of the 21st December 1933, and amongst the outstanding papers of interest were Dr. BISWAS'S (Calcutta) notes on a few cases of epidermoid carcinoma, a paper by Capt. N. N. ROY (Calcutta) on septic thrombosis of the cavernous sinus and orbital cellulitis, the treatment of after-cataract by Dr. N. CHATTERJI (Calcutta) which produced a lively discussion in which many members took part. Dr. J. N. BANERJI (Calcutta) read a comprehensive paper on School myopia in Bengal.

The Annual General Meeting of the All-India Ophthalmological Society took place on the evening of the 21st December at which a resolution advising more thorough legislation on the proper examination of the eye sight of drivers of motor vehicles was moved by Dr. NARAYAN RAO and carried unanimously. It was also resolved to draw the attention of some of the Universities in India to the absence of a special paper in ophthalmology in the final examination in medicine.

The following office-bearers were elected :

President—Lt.-Col. J. N. Duggan, C.I.E.
Vice Presidents—Lt.-Col. Wright, C.I.E., I.M.S.
Lt.-Col. E. O'G. Kirwan, I.M.S.
Secretaries—Dr. G. Zachariah, D.O.M.S.
Dr. B. N. Bhaduri.
Treasurers—Dr. S. N. Shroff, D.O.M.S.
Dr. K. N. Karanjia, F.R.C.S.

It was decided that the next meeting of the All-India Ophthalmological Society should take place during the Easter week of 1935 in Madras, and the subject for discussion should be Nutritional Disorders of the eye.

The congress was finally brought to a close on the night of the 21st December when it ended with a dinner at the Great Eastern Hotel at which many delegates and members were present, and the general impression was that the congress was a great success, and has led to much mutual understanding and co-ordination not only in the working out of scientific problems, but in bringing together workers from various parts of India.

Book-Review

The Medicinal and Poisonous Plants of Southern Africa

JOHN MITCHELL WATT, M.B., Ch.B. (Edin.) AND MARIA GERDINA BREYER-BRANDWIJK

(*E. & S. Livingstone, Edinburgh*)

This is a novel publication reflecting great credit, research and scientific spirit of the authors who have spared no pains to make the work a success. It is for the first time that a complete work giving all the available informations on the medicinal uses, chemical composition and pharmacological effects together with toxicology of the flora of Southern Africa has been placed before the profession. Apart from its special value in Southern Africa, it is of interest to every pharmacologist all over the world.

The book is well illustrated with some beautiful colour plates and diagrams, and deserves to be carefully read by all interested in the development of the drug industry on a scientific line.

B. N. G.

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Printed and published by Dr. Girin Mukherji from 67, Dharamtala Street, Calcutta
Printed at the USHA PRESS, 74, Dharamtala Street, Calcutta